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USE OF RADIUM IN TREATMENT OF DEAFNESS BY IRRADIATION

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The treatment of deafness with radon was first suggested by Crowe and Baylor [1] in 1939. The finding of large numbers of children with hearing defects in the high frequencies and the fact that many of these children had remarkable return of hearing after tonsillectomy and adenoidectomy led them to believe that these high frequency defects were due not to nerve defect, as was commonly thought, but rather to subclinical otitis media and eustachian salpingitis. They expressed the opinion that it was probable that overgrowth of lymphoid tissue in and about the eustachian tube was responsible, especially as many of the children had had previous and adequate adenoidectomy. Nasopharyngoscopic examination revealed that such a condition existed, but, obviously, it would be impossible to correct this by operation. They therefore devised a method of irradiating the eustachian orifice with radon, which was successful.

The difficulty with the treatment as described by Crowe and Baylor [1] is the fact that radon as they employed it is not easily available to many otologists. We, therefore, have designed simple appropriate instruments and established a technic which is within the reach of most clinics and which enables us to irradiate efficiently the lymphoid tissue in the region of the orifice of the eustachian tube with as little as 25 mg. of radium.

METHOD

Instruments.—The applicator consists of three parts (fig. 1): the handle, *A;* the cap, *B,* and the head, *C.* The handle is composed of a heavy, semiflexible steel

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1. Crowe, S. J., and Baylor, J. W.: Prevention of Deafness, J. A. M. A. **112**:585-590 (Feb. 18) 1939.

wire 13.4 cm. long, which is threaded at one end. The cap holds two 12.5 mg. radium needles in the head. It is an oval piece of brass 1 mm. thick and of sufficient size to cover the proximal and open end of the two chambers in the head of the applicator. There is a hole in one end of the cap just large enough to allow passage of the threaded end of the handle. On the under side and at the opposite end of the cap is a small steel plug, which fits snugly into the open end of one of the radium chambers of the head to prevent displacement of the radium needle. The head is constructed of brass 1 mm. thick. It is 3 cm. long, 0.6 cm. wide and

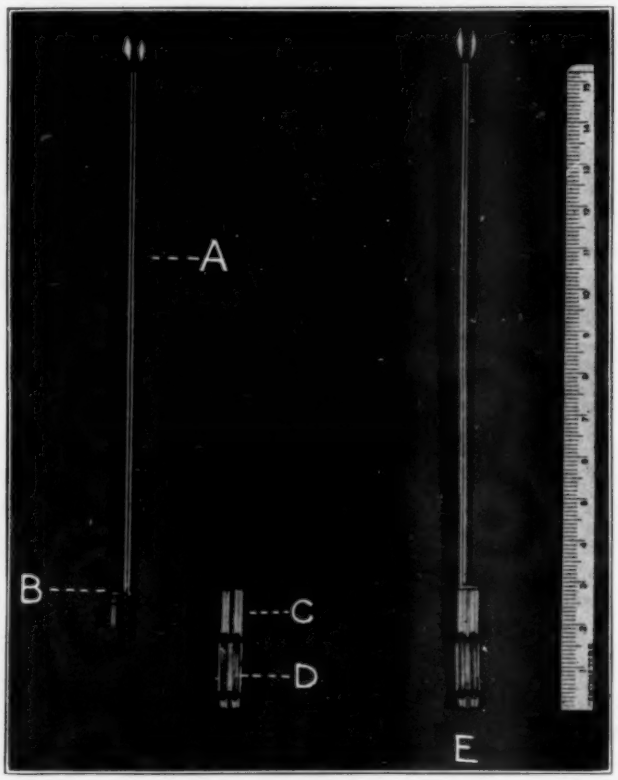

Fig. 1.—The radium applicators used, one being the mirror image of the other. One instrument has been assembled.

2.5 mm. thick. It has two parallel chambers, each of which accommodates one 12.5 mg. radium needle. The open end of one chamber is threaded to receive the threaded end of the handle. Thus, the threaded end of the handle holds one needle securely in place, and the plug retains the other needle in its respective chamber. In one side of each chamber of the head is an oval window 1.5 cm. long and the width of the chamber (fig. 1 *D*). This window permits the use of the radium without additional filter other than the wall of the needle. Figure 1 *E* shows the applicator with the parts assembled. Both applicators are loaded with radium in the radium room and are transported to the clinic in a long-handled lead carrier, the walls of which are 1 inch (2.54 cm.) thick (fig. 2).

Each needle is 20.3 mm. in length, with an external diameter of 1.35 mm. The needle is an alloy having a density of 8.7, and the wall is 0.3 mm. thick. The wall transmits 99.2 per cent of the gamma rays. Of the total amount of the radiation emanating from such a needle, approximately 35 per cent are beta rays.

In order to save time in the treatment of the patients, we have constructed two applicators, one the mirror image of the other. This requires a total of 50 mg. of radium but permits the treatment of both eustachian tubes in twelve minutes after local anesthesia has been established.

Previous experience with these needles in contact with the mucous membrane of the lip in children has established the erythema dose to be between 12 and 15 milligram hours. It was felt that approximately one third of an erythema dose (5 milligram hours) would be sufficient to cause regression of the hyperplastic lymphoid tissue. A twelve minute application delivers 5 milligram hours, as each applicator when loaded contains 25 mg. of radium. Such a short treatment time

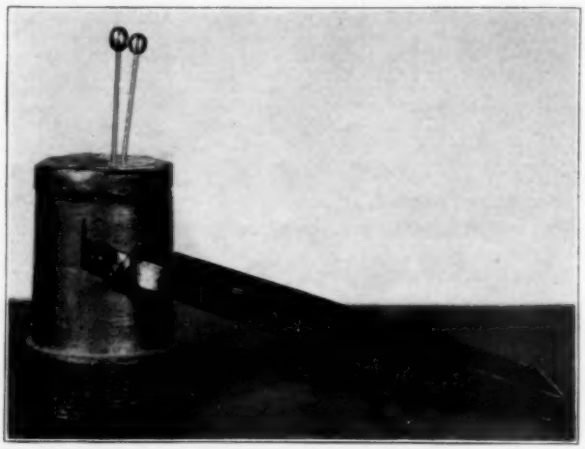

Fig. 2.—The lead carrier in which the radium is carried from the vault to the otologic clinic.

with this small amount of radium is made possible by the fact that we utilize a high proportion of beta radiation in actual contact with the area to be treated.

Technic.—Our procedure in the treatment of the patient is, first, to give careful attention to the cleaning up of all preexisting disease of the upper respiratory tract, including tonsillectomy and adenoidectomy, if this has not previously been done. After this, the patient is examined with the nasopharyngoscope under pontocaine anesthesia to determine the state of the os of the eustachian tube as regards hypertrophic lymphoid tissue. If there is extensive lymphoid hypertrophy about the tube or if the orifice seems swollen or edematous, the patient is given radiation and examined two weeks later to see how much the lymphoid tissue has regressed. The hearing is again checked at the end of a month and again in six months. In the majority of patients the tube will appear normal and open by the end of two to four weeks, and the hearing curve will be seen to approach normal. Not all patients will have a return to the normal curve, but they will show a partial response. Some will need further irradiation. So far as we have followed our patients, none has had a drop in the hearing level, and the regained hearing has been permanent. We arbitrarily allow at least three months between irradiation

treatments, as we have found that when the improvement is not as marked in two to four weeks as we should expect some patients will at the end of a three month period show a satisfactory response, not only in the hearing curve but in the regression of the lymphoid hypertrophy about the eustachian tube.

The technic of irradiation is easily carried out with the use of pontocaine anesthesia. Usually the instrument can be passed after the nasopharyngoscopic examination, without further medication. It is inserted through the inferior meatus, with the window of the carrier toward the lateral wall, and will drop into the

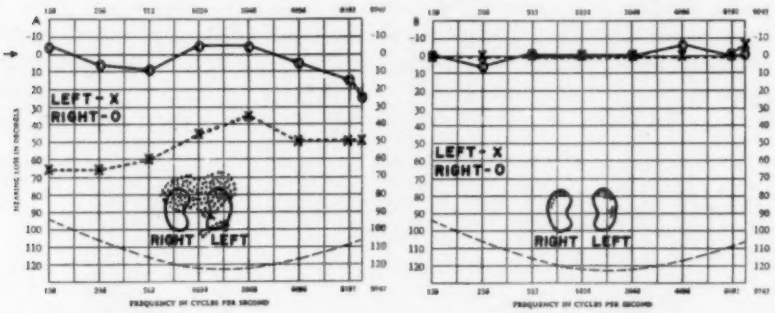

Fig. 3 (case 1).—(*A*) Audiogram for C. H., showing marked defect in the left ear. The schematic drawing shows the eustachian tube previous to treatment.

(*B*) Excellent return of hearing. Again, the eustachian orifices are schematically represented to show the reduction in hypertrophic lymphoid tissue.

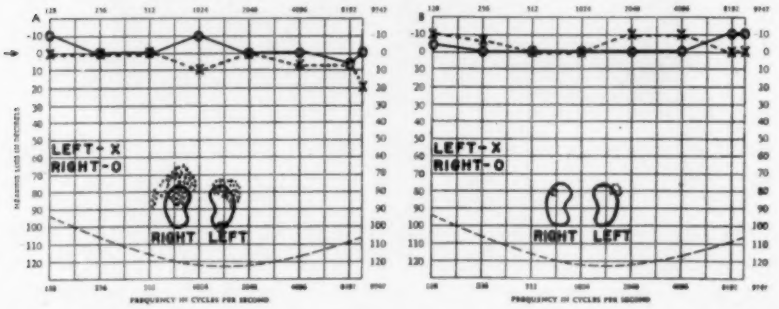

Fig. 4 (case 2).—(*A*) Audiogram for W. R., showing small hearing defect but large amounts of lymphoid tissue.

(*B*) One month later hearing is normal and the eustachian tubes are clear.

correct position and remain fixed there, owing to the shape of the radium carrier itself.

REPORT OF CASES

Six cases have been picked at random from the group of almost 100 which were selected for this technic. The history of deafness and the presence of eustachian adenopathy were the two main criteria for the selection of these cases. The 6 cases illustrate the average variations encountered. The hearing range was tested by the usual

audiometer equipment (6 B) available in most clinics; while this does not have a wide range, it is sufficient for detecting appreciable losses of hearing and indicating their recovery.

CASE 1.—C. H., an 11 year old white boy, was first seen at Strong Memorial Hospital on March 16, 1941, because of inattention in school. A history of repeated attacks of otitis media and a simple mastoidectomy in 1932 was obtained. His tonsils and adenoids had been removed in 1932. Examination revealed a scarred left drum and absence of tonsils. Nasopharyngoscopic study showed large amounts of lymphoid tissue about both eustachian tubes, represented schematically in figure 3. The audiogram also showed pronounced defect in hearing in the left ear. A 5

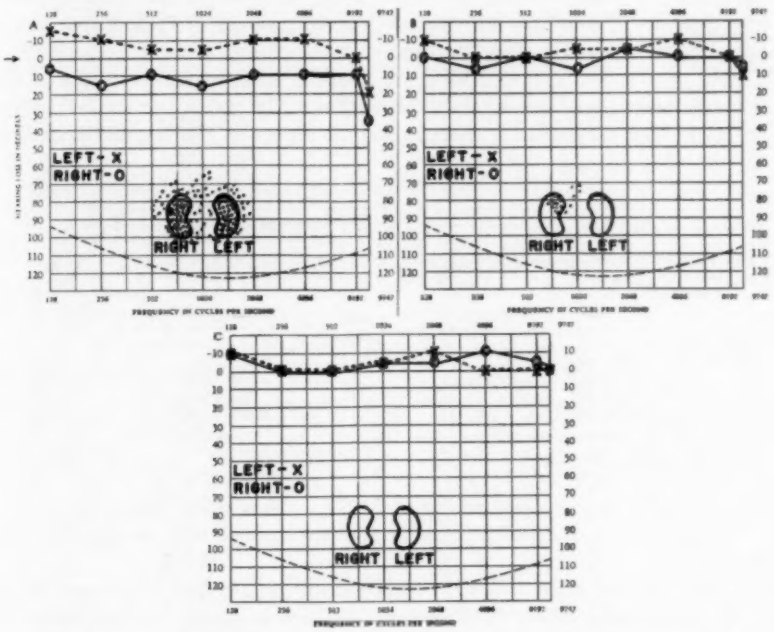

Fig. 5 (case 3).—(*A*) Audiogram for L. Q., showing moderately advanced deafness and profuse lymphoid hypertrophy.

(*B*) Two months after treatment some defect persists in the right ear, and lymphoid tissue is still present about the right eustachian orifice.

(*C*) By the end of three months both ears are normal and the lymphoid tissue has entirely receded.

milligram hour dose of radium was applied by the intranasal route. Reexamination of the patient's hearing one month later showed a truly remarkable response, and the eustachian tubes were clearly open. The patient voluntarily announced that he no longer received punishment at school for inattention.

CASE 2.—W. R., an 11 year old white boy, was first seen at Strong Memorial Hospital because his teacher had noted intermittent periods when the hearing in his left ear appeared defective. A history of repeated attacks of otitis media in childhood was obtained. Tonsillectomy and adenoidectomy were performed in 1936. Examination showed a scarred left drum and slight dulness of the right

drum. The nose and sinuses were normal. The tonsils had been cleanly removed. Bone conduction was greater than air conduction bilaterally, and the reaction to the Weber test was referred to the left. Nasopharyngoscopic examination revealed considerable adenoid tissue about the eustachian tubes bilaterally. On the basis of this finding, plus a slight upper tone defect and the history of intermittent difficulty in hearing, a 5 milligram hour dose of radium was given on April 30, 1941. A subsequent audiogram and nasopharyngoscopic examination made on May 30, 1941 showed satisfactory response.

Case 3.—L. Q., a 13 year old white boy, made his first visit to the otologic clinic of the Strong Memorial Hospital on Nov. 13, 1940 because of difficulty in hearing at school for several years. He gave a long history of repeated attacks of otitis media and infections of the upper respiratory tract. An adenoidectomy had been done in 1932, and the adenoids were again removed in 1934, during tonsillectomy. Examination revealed scarring of both drums, without perforation, a septal spur on the left and sprinkling of adenoid tissue about the vault of the nasopharynx, especially about the eustachian tubes. The audiogram showed a high tone defect in both ears, more pronounced on the right. A 5 milligram hour dose

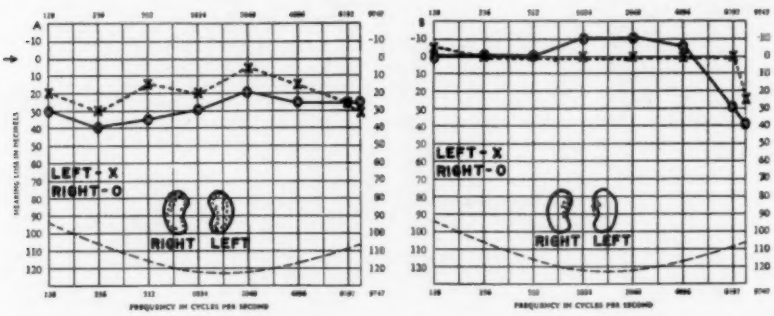

Fig. 6 (case 4).—(*A*) Audiogram for D. B., showing a marked defect in both ears. The amount of lymphoid tissue is less than usual with a defect of this magnitude.

(*B*) Five months after treatment improvement in hearing is shown for all except the high frequencies. A little lymphoid tissue is still present about the orifices. A second dose of radium is to be given.

of radium was applied by the intranasal route on November 13. By Jan. 15, 1941 only a slight defect for high tones remained, and in a final check on April 29, 1941 the hearing had returned to the normal level and both eustachian tubes were clear of hypertrophic lymphoid tissue. This case demonstrates how in some patients the full response may require several months.

Case 4.—D. B., an 11 year old white boy, had a history of deafness following a severe attack of measles about one year prior to his first visit to the Strong Memorial Hospital. He had had many attacks of otitis media before the measles, but no hearing loss had ever been noted subsequently. The family history was without significance and there was no history of allergy. Examination showed scarring of the drums with perforation bilaterally and absence of the tonsils, and nasopharyngoscopic inspection revealed moderate hypertrophy of the lymphoid tissue about the eustachian tubes. A 5 milligram hour dose of radium was applied bilaterally by the intranasal route on Sept. 18, 1940. By Feb. 11, 1941 he had had

an excellent rise in hearing level, aided in the last two months by nicotinic acid therapy. The defect in high tones shown in the last audiogram is indication that further radium therapy is in order.

CASE 5.—S. R., a 13 year old white boy, was referred to the otologic department of Strong Memorial Hospital by his school because of deafness of unknown duration, of which both the patient and the parents were unaware. His tonsils had been removed three years previously. Examination, except the nasopharyngoscopic, gave entirely normal results. Nasopharyngoscopic study showed heavy lymphoid

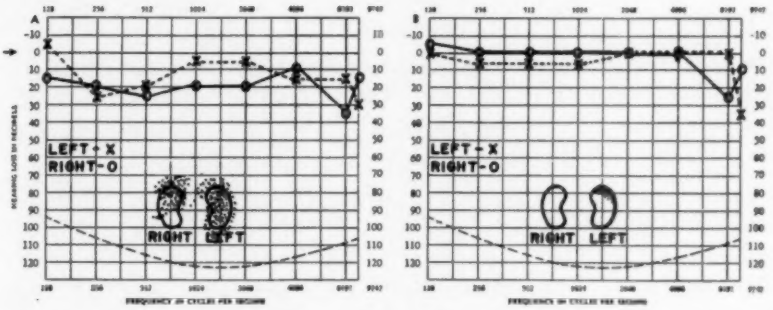

Fig. 7 (case 5).—(*A*) Audiogram for S. R., showing a marked hearing defect in both ears. There was profuse distribution of lymphoid tissue about the eustachian orifices, especially the left.

(*B*) After the second dose of radium the eustachian tubes appeared clean and the return of hearing was good except for the high frequencies.

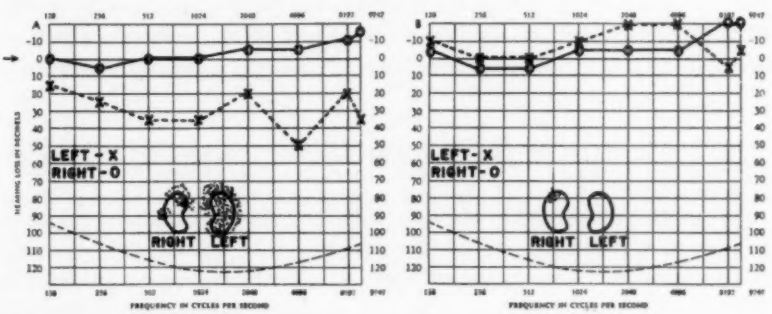

Fig. 8 (case 6).—Audiogram for R. L., showing marked hearing defect on the left and profuse lymphoid tissue about the eustachian tubes, especially the left.

((*B*) Three months later the patient showed a truly remarkable response in hearing and the eustachian tubes were clean.

involvement of both eustachian openings. A 5 milligram hour dose of radium was applied bilaterally by the intranasal route on Dec. 4, 1940. By April 29, 1941 he had regained most of his hearing, especially in the conversational ranges and his school difficulties had ceased. Because he still exhibited a marked defect for high tones and because the eustachian tubes still showed some lymphoid tissue, a second 5 milligram hour dose of radium was applied; subsequent improvement was noted

on June 4, 1941, at which time both eustachian tubes appeared clean on nasopharyngoscopic inspection.

CASE 6.—R. L., an 18 year old white girl, was first seen at Strong Memorial Hospital on May 21, 1941, when a history of difficulty in hearing for three to five years was obtained. A hearing test at school revealed a 30 per cent loss. Examination of the nose and throat showed normal ear drums and clean removal of tonsils, but a large amount of adenoid tissue about the eustachian orifices, especially on the left. A 5 milligram hour dose of radium was given bilaterally. Three months later the eustachian tubes were clean and hearing had returned practically to normal.

COMMENT

It is of interest to note in the cases cited that the onset of deafness was insidious and the otitis media and salpingitis remained subclinical over long periods. It becomes evident that the only method of detecting hearing defects in children with eustachian adenopathy is by use of the audiometer. Certainly, children with tonsils and adenoids still in place should be carefully checked, especially if they are having difficulty in school.

Irradiation of eustachian tubes overgrown with lymphoid tissue allows one to carry out treatment, with good results, in many cases. The return of hearing may be complete in some cases; at least one may control the process, preventing serious deafness in adult life.

Some difficulty may be experienced at first in selecting the patients, but as the examiner becomes more adept in combining the history and the nasopharyngoscopic findings the percentage of good results will increase. It must be remembered that in cases of advanced deafness a small return of hearing, or even the arrest of the process, should be considered a good result, especially as the patient enters the teens.

CONCLUSIONS

A simple effective method of irradiating the eustachian orifices with radium is presented.

The method renders excellent results in treatment of deafness secondary to hypertrophy of lymphoid tissue about the eustachian orifices once the examiner has acquired the necessary experience in selecting cases by combining the data from the history, the audiogram and the nasopharyngoscopic examination.

Care must be taken to eliminate any preexisting infection of the upper respiratory tract, tonsillectomy and adenoidectomy being performed when indicated.

This type of deafness is insidious in its onset and progress in the earlier stages and can be detected only by the wide use of the audiometer.

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LABYRINTHINE DROPSY AND MÉNIÈRE'S DISEASE

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The association of labyrinthine dropsy (hydrops labyrinthi; dilatation of endolymphatic spaces) with the clinical entity known as Ménière's disease was first described by Hallpike and Cairns [1] in 2 cases. Since then additional cases of labyrinthine dropsy and clinical Ménière's disease have been described by other investigators (Yamakawa,[2] 1 case; Hallpike and Wright,[3] 1 case, and Rollin,[4] 2 cases). In addition, Rollin described the same pathologic entity in 2 cases of deafness without vertigo and in 1 case without any available history.

The discovery of the dropsy, or dilatation of the endolymphatic spaces, provides a rational basis for the clinical syndrome, but neither the details of how the disturbance of function is produced nor the cause of the pathologic condition has as yet been satisfactorily explained.

The disease is now recognized as fairly common, but since it does not shorten life and the distressing symptoms tend to be transient, pathologic material has rarely been obtained, and then usually coincidentally.

The purpose of this report is to present the pathologic observations in a case of advanced bilateral deafness, associated with an extreme degree of labyrinthine dropsy of idiopathic origin.

REPORT OF A CASE

History.—On March 26, 1932 J. G., a man aged 67, came to the hospital because of attacks of dyspnea, which had recently become severe. A diagnosis of arteriosclerosis, myocardial insufficiency and hypertension was made. The blood pressure was 220 systolic and 160 diastolic. The medical record stated that he was "quite deaf" and had a history of noises in the ears and increasing deafness for the past fifteen years.

He was again admitted to the hospital on Oct. 18, 1932. At that time the blood pressure was 210 systolic and 140 diastolic. The medical record stated that

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From the Division of Otolaryngology of the University of Chicago.

1. Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol. **53**:625-654 (Oct.) 1938.

2. Yamakawa: Ztschr. f. Oto-Rhino-Laryng. **44**:181, 1938; cited by Rollin.[4]

3. Hallpike, C. S., and Wright, A. J.: Histologic Changes in Temporal Bones of Case of Ménière's Disease, J. Laryng. & Otol. **55**:59-66 (Jan.) 1940.

4. Rollin, H.: Zur Kenntnis des Labyrinthhydrops und des durch ihn bedingten Ménière, Hals-, Nasen- u. Ohrenarzt (Teil 1) **31**:73-109 (March) 1940.

"the patient was deaf in the right ear and could hear only a very loud voice in the left." Death occurred on October 23, from myocardial insufficiency. Autopsy was performed twenty-six hours later. The temporal bones were removed and placed in a 20 per cent concentration of degassed solution of formaldehyde U. S. P.

Contact could not be established with relatives, and attempts to obtain a more detailed history pertaining to the otologic condition were unsuccessful.

Pathologic Changes.—Right Temporal Bone: The mastoid region was normally pneumatized. Some air cells were present in the infralabyrinthine area and the inferior part of the apex. The labyrinthine capsule was normal. The fossula ante fenestrum and fossula post fenestrum were present. A few thin bands of adhesions were observed in the window niches. Otherwise the mucosa of the tympanum was normal.

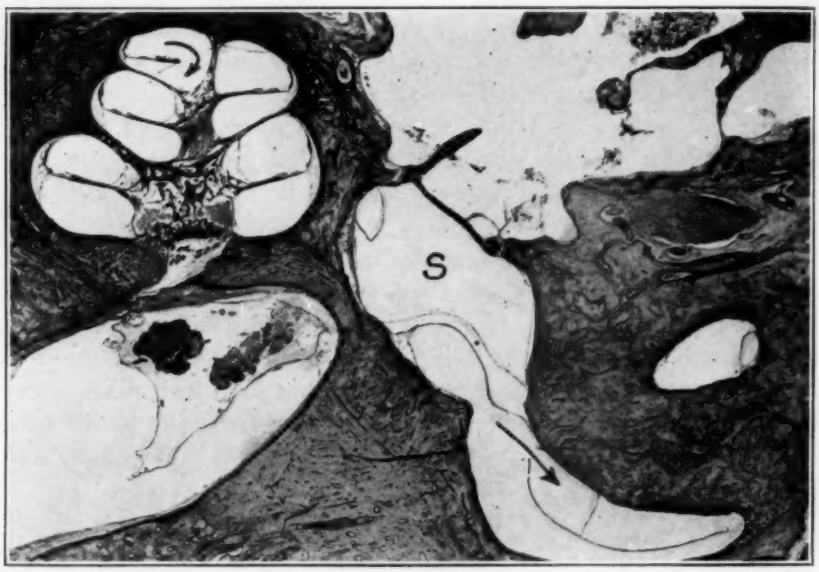

Fig. 1.—Section through the right labyrinth, showing the extreme dilatation of the ductus cochlearis and the saccule (*S*). The herniations of the ductus through the helicotrema and of the utricular wall into the common crus are denoted by arrows.

The membranes of the internal ear were well preserved. No air bubbles were observed.

Cochlea: The ductus cochlearis was dilated throughout its length to the extent that the scala vestibuli was limited to a small channel along the modiolar wall. The scala vestibuli decreased in size until it appeared obliterated near the helicotrema (fig. 1). The ductus cochlearis was herniated through the helicotrema into the scala tympani of the upper coil. Many trabeculae were seen in the remaining portion of the scala vestibuli. The cochlear aqueduct appeared normal.

The spiral ganglion showed some degeneration in the upper and lower regions, but both the ganglion and the associated nerve fibers were well preserved in the

middle coil and the upper half of the lower coil and must be considered as within the average range for the age period of the patient. Corti's organ was well preserved except for the hair cells. These showed extensive degeneration, which was probably explained by the period of twenty-six hours after death which preceded fixation.

The stria vascularis in some areas showed marked changes of a degenerative nature but in most areas appeared normal.

Vestibule and Semicircular Canals: The vestibule was almost completely filled by the dilated saccule and utricle. The walls of the saccule were dilated, with the exception of the reenforced area described by Perlman,[5] which extended from the outer border of the macula, and the outer saccular wall overlay the foot plate of the stapes. The utricle presented a striking appearance. The macula remained

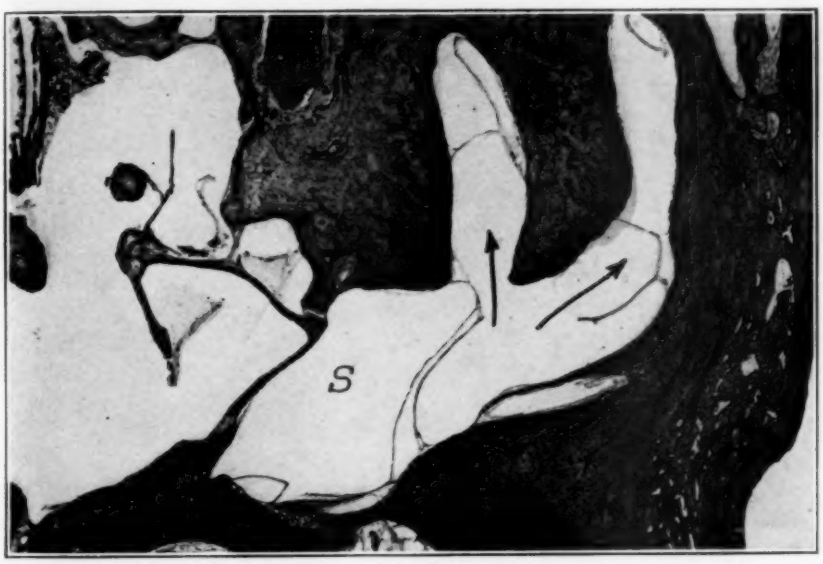

Fig. 2.—Dilated saccule (*S*) with the wall in contact with the foot plate of the stapes. The reenforced area of the saccular wall described by Perlman is shown. Herniations of the utricle into the perilymphatic space of the small end of the horizontal canal and the opening of the common crus are denoted by arrows.

in position, and the opposite wall was in contact with the bony wall of the vestibule. At the openings of the common crus and the small end of the horizontal canal into the utricle, the utricular wall, being much thinner than the membranous canal, had herniated into and filled the perilymphatic space of each of the canals for some distance (fig. 2). The membranous canal was not distorted.

At the ampullar ends of the posterior and horizontal canals some degree of herniation had also occurred. In both cases the thin utricular wall had been ballooned outward and had herniated between the wall of the ampulla and the

5. Perlman, H. B.: The Saccule: Observations on a Differentiated Reenforced Area of the Saccular Wall in Man, Arch. Otolaryng. **32**:678-691 (Oct.) 1940.

bone. The membranous wall of the ampulla, being of a thickness corresponding to that of the membranous canal, was thereby left with the appearance of projecting into the lumen of the utricle (fig. 3).

In the case of the posterior canal the herniated portion of the utricular wall had extended beyond the ampulla into the perilymphatic space of the canal. In so doing displacement and distortion of the membranous ampullar wall had been produced (figs. 3 and 4). In the case of the horizontal canal, the herniation did not extend beyond the ampulla but the ampullar wall appeared to project into the utricle, with the herniated utricular wall covering its posterior aspect. Some distortion of the ampullar wall was evident, but to a much less extent than in the case of the posterior canal.

Fig. 3.—Section through the ampulla of the posterior vertical canal, showing the herniation of the utricle (arrow) between the bone and the wall of the ampulla (*b*), which is thereby distorted and deprived of support in much of its extent. The herniated utricular wall is seen as it is reflected from the outer surface of the membranous ampullar wall along the bone.

The membranous canals had relatively thick walls and appeared normal in contour except for the distortion at the points of entry into the utricle previously described.

Endolymphatic aqueduct and sac: There appeared to be some dilatation of the central portion of the aqueduct. The lining seemed to have fewer folds and convolutions than might be considered normal. The surrounding connective tissue appeared fairly dense, with fewer dilated capillaries than usual. The epithelial lining seemed to be in good condition, with the exception of one or two areas where it was poorly preserved (fig. 5).

The endolymph stained pink in the interstices of the aqueduct, as well as in the channel between the tectorial membrane, Corti's organ and the spiral lamina.

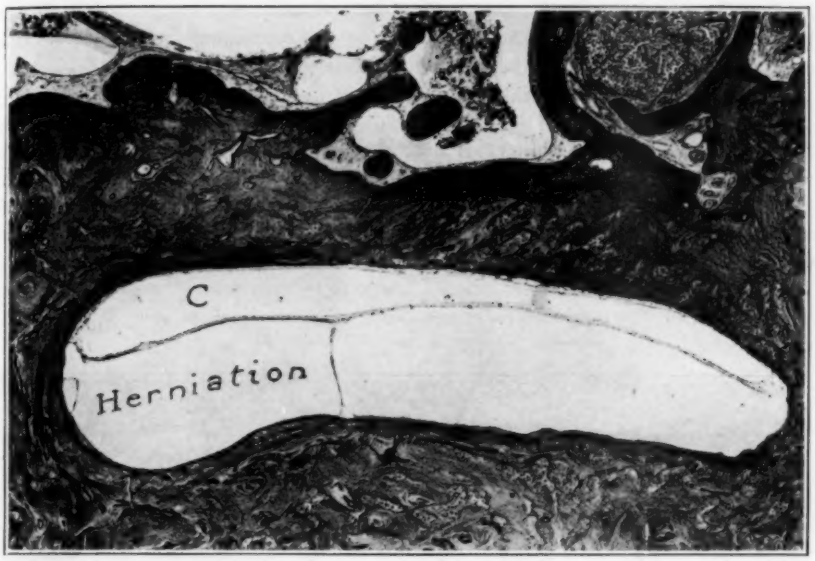

Fig. 4.—Section through the posterior canal beyond the ampulla (present in fig. 3), showing the membranous canal (*C*) and the herniation of the utricle, which has extended through the ampulla and completely fills the perilymphatic space for some distance.

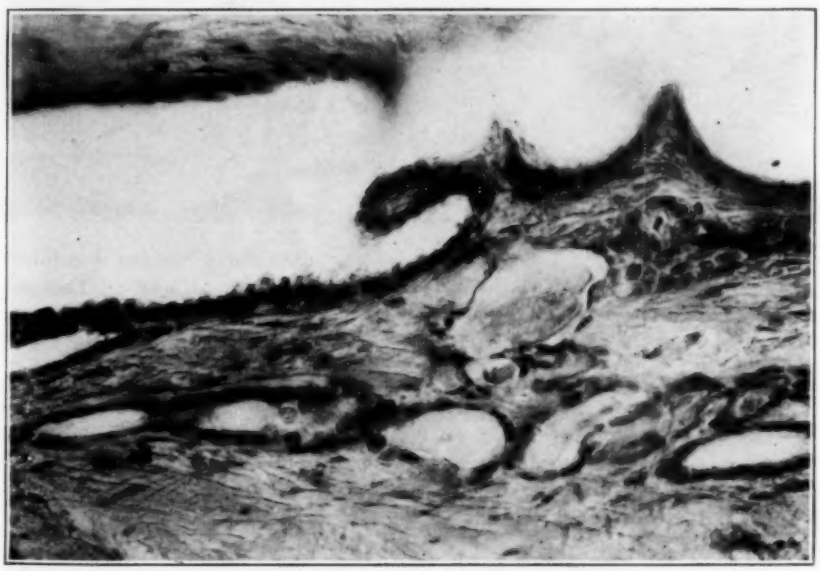

Fig. 5.—High power (× 387) magnification of a portion of the endolymphatic aqueduct on the right side. The epithelial lining appears to be in fair condition. The folds of epithelium and the capillaries may be less numerous than normal.

Left Temporal Bone: Pneumatization was normal and corresponded in degree to that on the right side. The mucosa lining the tympanum and the connecting air spaces was normal. The labyrinthine capsule was also normal.

Cochlea: The ductus cochlearis was dilated to a degree corresponding to that on the right side (fig. 6). The scala vestibuli was reduced to a trabecula-containing channel, which appeared obliterated near the helicotrema. The ductus cochlearis was herniated into the scala tympani of the upper coil. The stria vascularis was degenerated in some areas but was normal throughout most of its extent. Corti's organ and the tectorial membrane were in good condition except for extensive degeneration of hair cells and corresponded to the structures on the opposite side. The spiral ganglion and the associated fibers were in a fair state of preservation for a man of 67, the condition corresponding to that on the right side.

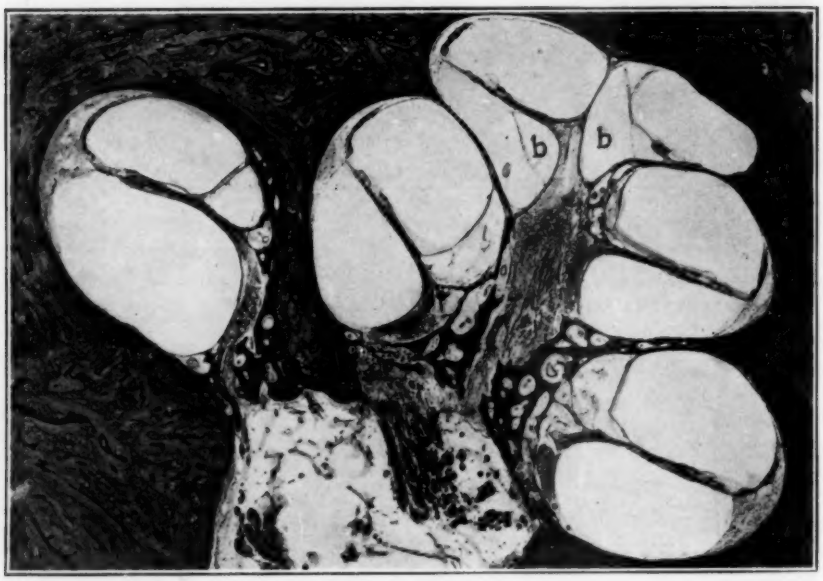

Fig. 6.—Section of the cochlea of the left ear. The ductus cochlearis is dilated to an extreme degree and has herniated into the scala tympani at *b*. The scala vestibuli is progressively reduced in size from the basal coil upward and appears to be obliterated in the upper coil.

Vestibule and semicircular canals: The utricle and saccule were dilated, but to a somewhat lesser degree than on the right side (fig. 7). The saccular wall lay in apposition to the foot plate of the stapes. There was beginning herniation of the utricular wall at the ampulla of the posterior canal (fig. 8) and at the ampulla and at the openings of the common (fig. 9) and the small crus of the horizontal canal.

Endolymphatic aqueduct: The aqueduct was dilated to some extent. The epithelium was well preserved and the convolutions appeared of normal extent. The surrounding connective tissue showed no definite abnormality, and the capillary supply also seemed to be normally abundant. The endolymph stained pink with

hematoxylin and eosin and contained about the normal number of cells. The aqueduct on this side appeared more normal than that on the opposite side, but the difference in the extent of hydrops was slight (fig. 10).

Summary of Pathologic Changes.—Right Ear: Cochlea: The ductus cochlearis was extremely dilated at the expense of the scala vestibuli. Corti's organ and the nerve elements were normally preserved except for the hair cells, which showed extensive degeneration. The stria vascularis showed limited degeneration.

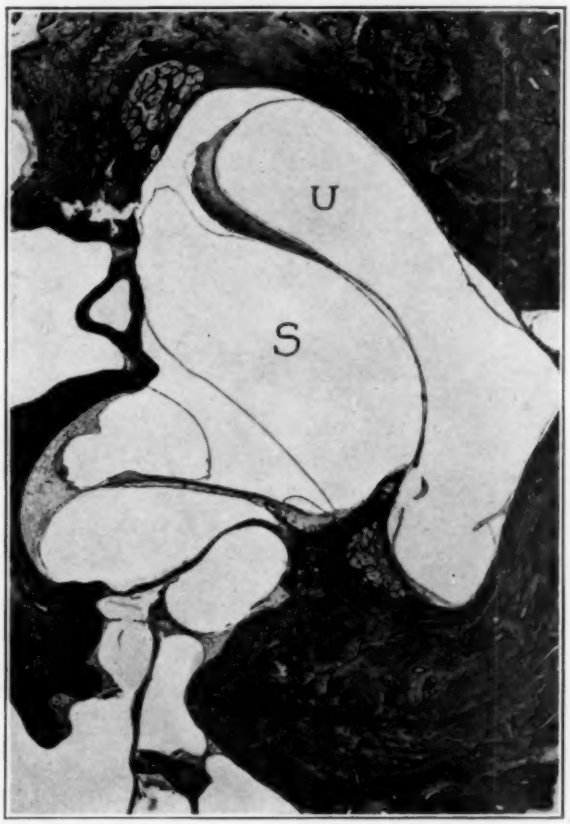

Fig. 7.—Section through the vestibule of the left ear. The saccule (*S*) and the utricle (*U*) almost fill all available space. The saccule is in opposition to the foot plate of the stapes.

Vestibule and semicircular canals: There was extensive dilatation of the saccule, which overlay the foot plate of the stapes. The utricle was dilated and had herniated into the small ends of the semicircular canals, so that the utricular wall lay alongside the membranous walls of the canals, and had also herniated at the ampullae of the posterior vertical and horizontal canals in such a way as to distort and displace the wall of the ampulla opposing the cupula and crista.

Endolymphatic aqueduct and sac: Dilatation was moderate. The lining epithelium in the central part was well preserved. The convolutions and the surrounding connective tissue were somewhat reduced.

Left Ear: The pathologic changes in this ear corresponded closely to those in the right, but the dilatation of the saccule and the herniations of the utricle were considerably less extensive.

Fig. 8.—Section through the vestibule and ampulla of the posterior canal of the left ear. *S* indicates the dilated saccule; *U*, the utricle, and *P*, the ampulla of the posterior canal. Beginning herniation of the thin utricular wall between the thick wall of the ampulla and the bone is indicated by the arrows.

CORRELATION OF THE PATHOLOGIC AND THE CLINICAL SYNDROME

The 9 cases of labyrinthine dropsy of unknown cause described by previous authors indicate that the same pathologic condition in the inner ear may be found in cases of true Ménière's disease and in cases in which deafness of the same type is present but there is no history of attacks of vertigo. The case reported here belongs to the latter group.

It would appear therefore that this type of lesion of the inner ear may fail to produce vertigo and that the deafness and tinnitus are the only constant symptoms. It must be considered, however, that a characteristic of Ménière's disease is the frequent disappearance of the attacks of vertigo after a few years while the deafness continues or progresses. Also, in former years the occurrence of vertigo was inter-

Fig. 9.—Vertical section at the posterior border of the vestibule of the left ear. The extremity of the dilated saccule (*S*) and the edge of the utricular macula (*M*) are seen. The utricular walls are dilated, with small herniations at the ampulla of the horizontal canal (arrows), and a diffuse dilatation is seen in the end of the common crus (*CC*).

preted as a gastrointestinal symptom, and not as a disease of the ear or as a disturbance related to deafness. The possibility remains therefore that in cases of long-standing disturbance, such as the one described here, the full syndrome may have been present at an earlier stage.

The clinical characteristics of Ménière's disease were well known and the existence of some disturbance of the endolymphatic system suspected (Crowe [6]) before the association with labyrinthine dropsy was recognized. For a diagnosis of Ménière's disease auditory disturbances must be combined with intermittent attacks of vertigo which cannot be attributed to a known local or system disease.

The auditory involvement exhibits several distinctive characteristics. Deafness usually begins in the lower tones, and the characteristic wide fluctuations in the hearing threshold occur in this range. Bone conduction is usually decreased to a variable degree, but less than with

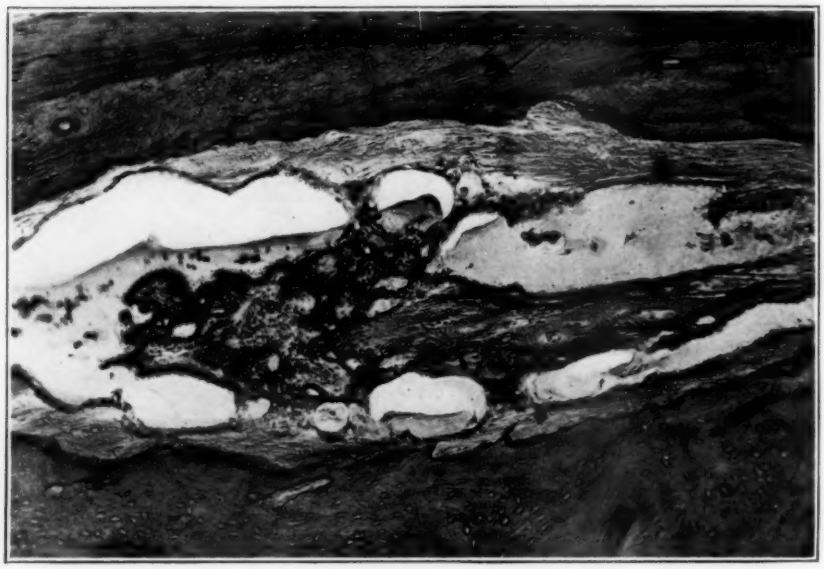

Fig. 10.—Section through the epithelial structure in the aqueductus endolymphaticus of the left ear.

lesions known to affect the nerve apparatus. Deafness fluctuates as much as 30 decibels or more in the various stages of an attack. The attack of vertigo usually follows a period of increasing deafness, tinnitus or sensation of blockage. Fluctuations in hearing may occur without an attack of vertigo. Disturbance of tone quality and of pitch is frequent and is commonly termed "diplacusis." Auditory symptoms are the most constant characteristic of the disease. When deafness has occurred it persists and may even progress after the stage of attacks of vertigo has passed.

6. Crowe, S. J.: Studies Based on Examination Made Before and After Intracranial Division of the Eighth Nerve, Medicine **17**:1-36 (Feb.) 1938.

Attacks of vertigo are commonly preceded by an aura of gradually or rapidly increasing auditory symptoms and may vary in duration, severity and frequency. Long periods may elapse between attacks, sometimes with recurrences after several years. Vestibular excitability, as indicated by caloric tests, remains normal in the early stages but decreases as the disease becomes advanced. The occurrence of attacks of vertigo or of postural vertigo, without any disturbances of the auditory apparatus, cannot be considered to constitute Ménière's disease, since there is no proof that the lesion lies in the peripheral vestibular apparatus.

The explanation has been offered that the disturbances of function produced by the labyrinthine dropsy have been caused by increased pressure of the endolymph acting directly on the sensory epithelium. However, this theory is untenable, for the reasons to be indicated.

Kobrak [7] showed that pressure changes in the cerebrospinal fluid in rabbits are transmitted to and are in equilibrium with the perilymph and that in the human middle ear pressures are transmitted to the perilymph. Since fluid is incompressible, any variation in pressure of the perilymph is transmitted to the endolymph and equilibrim is maintained.

In diseases, such as brain tumor, in which the cerebrospinal fluid pressure may increase several hundred millimeters of water, because these pressures are transmitted to the labyrinthine fluids the sensory epithelium is exposed to prolonged high pressures and to sudden wide variations in pressure during treatment. Yet in such conditions deafness or vertigo is not a characteristic of the wide variations in pressure.

Any increase in the volume of endolymph, on the other hand, will exert a pressure greater than that of the perilymph only to the extent necessary to dilate the thin membranes the infinitesimal amount necessary to establish pressure equilibrium. The perilymph, in turn, is in equilibrium with the cerebrospinal fluid. The pressure exerted on the sensory epithelium within the endolymphatic system can therefore exceed that of the cerebrospinal fluid only by the infinitesimal amount necessary to overcome elasticity of the limiting membranes sufficiently to maintain pressure equilibrium. Such pressures must be infinitesimal in comparison with pressure changes to which the labyrinthine fluids are known to be exposed without loss of function.

An increase in osmotic pressure due to an alteration of chemical constituents of the endolymph could account for labyrinthine dropsy. In such circumstances a state of pressure equilibrium would be reached and maintained as long as the possibility existed for further expansion

7. Kobrak, H.: Untersuchungen über den Labyrinthdruck, Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:456-463, 1933.

of the endolymphatic spaces at the expense of the perilymph. The pressure exerted on the sensory cells would, again, be infinitely less than that which frequently occurs in the cerebrospinal fluid, and consequently the labyrinth, without loss of hearing or disturbance of equilibrium.

The possibility of a toxic effect on the sensory epithelium, due to an alteration in the chemical constituents of the endolymph, must be recognized, but certain clinical and pathologic features tend to indicate a physical or mechanical explanation for the disturbances rather than a toxic effect. The state of preservation of the spiral ganglion and associated fibers in this case must be considered as commensurate with good function and does not explain the severe bilateral deafness. This would be consistent, also, with the persistence of a fair degree of bone conduction, such as is found in Ménière's disease (Mygind and Dederding [8]). The degeneration of hair cells in this case, with an otherwise normal Corti organ and tectorial membrane, could be explained on the basis of postmortem change, although some toxic effect during life could not be ruled out.

No evidence of rupture of the membranous labyrinth was present. Although the sections showed obliteration of the scala vestibuli in the upper coil of the cochlea by the dilated ductus cochlearis, this does not indicate that the walls were adherent. A potential space probably remained which would permit passage of perilymph and maintain communication between the scala tympani and the remaining perilymphatic spaces.

The more logical explanation for the deafness appears to be on a physical basis. Since definite proof of the method of sound analysis in the cochlea is lacking, the explanation of the auditory symptoms in this condition must be indefinite. It is known, however, by direct observation that the foot plate of the stapes is set in motion by the sound waves and that the column of perilymph in the cochlea must therefore also be set in motion. Whether or not von Bekesy's [9] interpretation of the method of stimulation of the hair cells is correct, it appears reasonable to suppose that the gross disturbance in the size relationship of the saccule, the cochlear duct and the scala vestibuli would result in interference with transmission of the wave impulse and reduction of intensity of the stimulus. Impairment of hearing by air conduction could thereby occur, but with a normal nerve apparatus bone conduction could persist to a greater degree than with nerve degeneration. Fluctuations in hearing would, then, be explainable on the basis of the temporary

8. Mygind, S. H., and Dederding, D.: Pathogenesis of Ménière's Disease and Kindred Diseases, Ann. Otol., Rhin. & Laryng. **47**:938-946 (Dec.) 1938.

9. von Bekesy: Zur Theorie des Hörens bei der Schallaufnahme durch Knochenleitung, Ann. d. Physik. **3**:111, 1932.

increase or decrease in dilatation of the ductus cochlearis. The occurrence of disturbances in pitch and quality, or diplacusis, can be explained on the same basis.

The investigations of Steinhausen,[10] repeated by Dohlman and others, have demonstrated the normal anatomic relation of the structures in the ampullae of the semicircular canals. Steinhausen showed that the cupula in life extends to the opposing ampullar wall and that any fluid displacement in the canal is accompanied by deviation of the cupula, which thereby stimulates the underlying hair cells. Any mechanical disturbance of an ampulla which would either produce a deviation of the cupula or prevent its normal movement in response to a normal stimulus would be sufficient to cause vestibular imbalance and produce vertigo and nystagmus.

The mechanism for the production of such a disturbance in equilibrium existed in the present case in the herniations of the utricular wall, which were present in both ears. On one side there was gross distortion of the ampullar wall of the posterior vertical canal (fig. 3) by the herniation, which had extended well beyond the ampulla, while at other ampullae distortion of a lesser degree was seen. Such displacement of the ampullar wall provided the basis for a disturbance of equilibrium.

The "aura" before an attack represents the period when dilatation, and therefore the tendency to herniate, is increasing. The attack may represent displacement of the wall of an ampulla and, consequently, either deviation of the cupula or interference with its movement.

The existence of dilatation of the endolymphatic spaces provides a rational explanation for the effectiveness of certain types of medical therapy. The volume of endolymph should be influenced by the same factors as those which alter the volume of cerebrospinal fluid. On this basis, the favorable effect of limitation of fluid intake, as pointed out by Mygind and Dederding, is explained, as well as the good results of treatment with salt-free diet and ammonium chloride or potassium salts.

ETIOLOGY

The histopathologic picture has not provided any conclusive evidence as to pathogenesis. Labyrinthine dropsy (hydrops labyrinthi) has been commonly observed in severe grades of serous labyrinthitis and has been produced experimentally by inflammatory or toxic irritation by Wittmaack.[11] Also, it occurs with such inflammatory lesions as syphilitic

10. Steinhausen, W.: Ueber den Nachweis der Bewegung der Cupula in der intaken Bogengangsampulle des Labyrinthes bei natürlichen rotatorischen und calorischen Reizung, Arch. f. d. ges. Physiol. **228**:322, 1931.

11. Wittmaack, K., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1925, vol. 12, p. 290.

osteitis and labyrinthitis, as well as in Paget's disease. It appears that the increase in endolymph in these conditions represents increased secretion in response to inflammatory or toxic irritation.

The perilymphatic spaces respond readily to inflammatory irritation or trauma by proliferation of connective tissue and formation of new bone.

In the present case and in most of the previously reported cases there was absence of any evidence of disease of the middle ear spaces or of the labyrinth capsule. There was also no evidence of inflammatory reaction in the perilymphatic spaces or of blockage in the cochlear aqueduct. The opinion was expressed by Hallpike and Rollin that some interference with absorption by the epithelial structure in the endolymphatic aqueduct is indicated by the histopathologic picture. On the basis of the histopathologic alterations in the present case, such an explanation might be considered tenable with respect to changes on the right side but scarcely with respect to those on the left.

Several theories as to the etiology have been advanced on the basis of clinical observation, the most common being a focus of infection, allergy or vasomotor instability. From a pathologic standpoint, the theory of a focus of infection appears to be untenable because of absence of any evidence of inflammatory reaction within the labyrinth. The roles of "allergy" and "vasomotor instability" appear to merit investigation. At present there is insufficient evidence for the assumption that allergy could produce such a disturbance in the endolymphatic system. Clinically there are strong contraindications. Allergic manifestations are rare in Ménière's disease, and attacks of vertigo are not usual in allergy, which is an extremely common condition. There is, however, strong clinical evidence that vasomotor instability may be the basis for attacks of vertigo in most cases of the true Ménière syndrome.

Further investigations of the chemical constituents of the endolymph and of the factors which may produce an increase or a decrease in its volume are necessary to an explanation of the pathogenesis of the disease.

SUMMARY

The pathologic changes in the inner ear in a case of idiopathic bilateral labythinthine dropsy (hydrops labyrinthi) with severe deafness is described.

From the cases reported to date the evidence indicates that labyrinthine dropsy of this type constitutes a pathologic entity which may produce either auditory disturbances alone or the clinical syndrome known as Ménière's disease.

The histopathologic changes in this case provide evidence to explain the interference with function in this disease. The functional dis-

turbances cannot be explained on the basis of increased endolymphatic pressure because of the existing mechanism to preserve pressure equilibrium with the perilymph and cerebrospinal fluid.

The auditory disturbances characteristic of this disease appear to be most adequately explained on a mechanical or physical basis. The distortion of the saccule and Reissner's membrane and the resulting disproportion of the scala media and the scala vestibuli, along with a relatively well preserved nerve apparatus, suggest an interference with sound transmission in the column of fluid in the cochlea as the cause of disturbed function.

The disturbance of vestibular function and the attacks of vertigo are explained on a physical basis by the occurrence of herniations from the utricle, invading the canals and producing distortion of the walls of the ampullae. Distortion of an ampulla interferes with normal function of the cupula ampullaris and explains both the attack of vertigo and the depression of caloric excitability in the later stages.

The histopathologic picture explains the favorable effect of certain types of treatment but fails to permit any conclusion as to the pathogenesis. Neither has clinical observation provided any definite indication of the etiologic factors in this type of disease of the inner ear.

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LIPOMA OF LEFT MAIN BRONCHUS

REPORT OF A CASE AND REVIEW OF LITERATURE

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Although malignant tumors of the tracheobronchial tree represent at least 10 per cent of all malignant growths, benign tumors in the air passages distal to the larynx are infrequently encountered. More general employment of bronchoscopy in diagnosis and treatment of chronic pulmonary disease has revealed a considerable number of benign tumors in the trachea and bronchi, but growths of this type are still sufficiently rare to justify placing cases on record. Of the benign tumors that have been observed, lipomas or fibrolipomas have constituted about 5 per cent.

The first case, reported by Feller,[1] was discovered post mortem in the bronchus of the middle lobe of the right lung of a woman 66 years of age. Myerson[2] reported a case of fibrolipoma in the left main bronchus of a man aged 47 and was able to effect a removal through the bronchoscope, with complete relief of symptoms. Honig[3] encountered a case post mortem in the left main bronchus of a man aged 54. Kernan[4] successfully removed a large lipoma from the right main bronchus at the bifurcation of the trachea in a man 50 years of age. Another case of fibrolipoma of the left main bronchus with successful removal was reported by McGlade.[5] Jackson and Jackson[6] mentioned a case of lipoma of a bronchus but did not record the details. Wessler

From the Medical College of Virginia.

1. Feller, A.: Ueber ein lipomähnliches Hämatom der Lunge, Virchows Arch. f. path. Anat. **236**:470-480, 1922.

2. Myerson, M. C.: Benign Neoplasms of the Bronchus: Report of a Case of Fibrolipoma of the Left Main Bronchus Removed Through the Bronchoscope, Am. J. M. Sc. **176**:720-726 (Nov.) 1928.

3. Honig, A.: Ein lipomartiges Gebilde des linken Stammbronchus, Monatschr. f. Ohrenh. **68**:155-167 (Feb.) 1934.

4. Kernan, J. D.: Three Unusual Endoscopic Cases, Laryngoscope **37**:62-64 (Jan.) 1927.

5. McGlade, T. H.: Fibrolipoma of the Bronchus: Report of a Case, Ann. Otol., Rhin. & Laryng. **48**:240-243 (March) 1939.

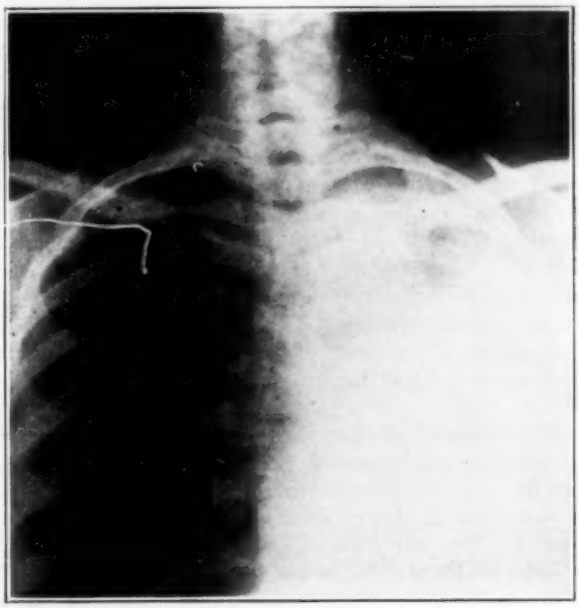
6. Jackson, C., and Jackson, C. L.: Benign Tumors of the Trachea and Bronchi, with Especial Reference to Tumor-Like Formations of Inflammatory Origin, J. A. M. A. **99**:1747-1754 (Nov. 19) 1932.

and Rabin [7] reported 17 cases of benign tumor of the bronchus, in 1 of which a fibrolipoma was present in the bronchus to the lower lobe of the left lung. This tumor was removed through the bronchoscope, with recovery of the patient.

The following report concerns what is apparently the eighth case of lipoma of the bronchus to be recorded.

REPORT OF A CASE

A Negress aged 29 was admitted to St. Philip Hospital for examination on March 6, 1940. She had been suffering for five years from cough, productive of



Roentgenogram of thorax, showing infiltration and atelectasis of the left lung secondary to obstruction from lipoma of the bronchus.

foul, purulent sputum, and periods of fever. She felt fairly well during the time when she was afebrile, but when her temperature was elevated she lost weight and strength and felt generally miserable. The bouts of fever were associated with chills and an increase in the amount of sputum. The last period of fever had begun in January 1940 and had continued, with a daily afternoon temperature of from 102 to 103 F. She had been confined to bed for two months prior to examination. Wheezing had been noted at times after a severe coughing spell.

Abnormal physical findings were limited essentially to the thorax and consisted of a dull to flat percussion note over the entire left side, with diminished breath

7. Wessler, H., and Rabin, C. B.: Benign Tumors of the Bronchus, Am. J. M. Sc. **183**:164-180 (Feb.) 1932.

sounds over the left lung, especially at the base posteriorly. There were many rales over the upper anterior portion of the left lung. Prior to our examination left pleural effusion had been considered and an aspirating needle had been inserted into the pleural space, but fluid was not obtained.

Hemoglobin was estimated at 72 per cent, with 15,100 leukocytes, 91 per cent of which were polymorphonuclear neutrophils. Other laboratory studies, including several examinations of the sputum for tubercle bacilli, disclosed nothing of significance.

Roentgenoscopic examination of the thorax showed deviation of the heart and mediastinum toward the left side and narrowing of the intercostal spaces, indicating atelectasis of the left lung. There were multiple small cavities between the first and the second rib anteriorly. The right lung was apparently free from abnormality (figure).

On March 8, bronchoscopic study revealed a tumor attached by a wide base to the anterior wall of the left main bronchus, proximal to the opening of the bronchus to the upper lobe. The bronchial lumen was occluded, but a small amount of pus exuded around the growth posteriorly. When the patient coughed, pressure of the bronchial wall on the mass prevented evacuation of secretion. The tumor was smooth, without evidence of ulceration, and was paler than the so-called bronchial adenoma, but it was thought most likely to be a benign adenoma. Tissue removed for microscopic examination proved the lesion to be a fatty tumor covered with normal epithelium. There was chronic inflammation of the submucosal portion.

Because of extensive disintegration of the lung distal to the tumor, pneumonectomy was recommended rather than removal of the growth through the bronchoscope. This method of treatment was further deemed advisable because of the broad base of attachment of the tumor to the bronchial wall and the tough consistency of the mucosal covering of the growth, making removal by snare or biting forceps difficult.

When the patient refused to have pneumonectomy, coagulation of the tumor through the bronchoscope was attempted. On March 12 the tumor was coagulated in two areas, and the treatment was repeated ten days later. At the second examination the tumor was reduced in size about one third, the lumen of the bronchus was much larger and pus was draining freely around the growth. Forty-eight hours after the second treatment the patient's temperature returned to normal, and four days later she was permitted to go home, with instructions to return in a month for reexamination. Further observation of the patient has not been possible, but communications from her physician indicate that she continues to suffer from suppuration in the lung.

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STUDIES ON PUPILS OF THE PENNSYLVANIA SCHOOL FOR THE DEAF

IV. MECHANISM OF INHERITANCE OF DEAFNESS

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It is well known that the majority of young children who are profoundly deaf are stated to have been born deaf, and it has long been accepted that this presumed congenital deafness is often hereditary. Before the development of the modern views on modes of inheritance teachers and other persons in contact with the so-called deaf mutes noted that in certain families the defect appeared generation after generation and that in others it frequently occurred among the offspring of consanguineous marriages (Bell [1]; Fay [2]). In recent years otologists familiar with genetic analysis have, from the study of a limited number of pedigrees, arrived at the conclusion that this form of profound deafness is transmitted according to a simple mendelian scheme (Albrecht [3]; Ciocco and associates [4]).

The implications of such a conclusion and its importance viewed either from a broad public health standpoint or from the position of the otologist whose advice is requested are not clearly perceived because of

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1. Bell, A. G.: Upon the Formation of a Deaf Variety of the Human Race, Mem. Nat. Acad. Sc. **2**:177-262, 1883.

2. Fay, E. A.: Marriages of the Deaf in America, Washington, D. C., Volta Bureau, 1898.

3. Albrecht, W.: Ueber die Vererbung der konstitutionell sporadischen Taubstummheit, der hereditären Labyrinth-schwerhörigkeit und der Otosclerose. Arch. f. Ohren-, Nasen- u. Kehlkopfh. **110**:15-48, 1922.

4. Ciocco, A.; Hughson, W., and Palmer, C. E.: Studies on Pupils of the Pennsylvania School for the Deaf: II. Resemblances in the Auditory Acuity of Siblings, Human Biol. **11**:259-268, 1939. Hughson, W.; Ciocco, A., and Palmer, C. E.: Studies on Pupils of the Pennsylvania School for the Deaf: I. Auditory Acuity, Arch. Otolaryng. **29**:401-416 (March) 1939.

the inherent complexity of the problem of deafness. There are many immediate causes of auditory impairment. It can come on at all ages and become manifest in several forms. Hence, a family history of deafness intrinsically has little meaning in terms of genetics unless the characteristics of the impairment are more or less specified. Conversely, the absence of any deafness in the immediate family of a deaf person does not constitute proof that the deafness in question is not inherited. Thus when it is desired, as in the present investigation, to reduce the problem of the type of profound deafness encountered in a school for the deaf to a level which will yield more easily to the application of the established principles of prevention and correction, the need is felt for a clearer definition of the criteria that will identify the form of deafness which is inherited. By the establishment of such criteria a more precise estimate of the frequency of inherited deafness among the cases of profound deafness in children may be obtained, and other factors which may play an important role in the occurrence of congenital deafness may be evaluated. Starting from this point of view, a survey of the families of the pupils of the Pennsylvania School for the Deaf was implemented to obtain information on (1) the incidence of deafness among the brothers and sisters, parents and grandparents of the children; (2) the pregnancy history of the mothers of the deaf children and their brothers and sisters, and (3) the mental and physical development of all the children. This paper is the first report on the survey and deals only with the familial concentration of deafness.

THE SURVEY

The resident student body of the Pennsylvania School for the Deaf numbers approximately 535 pupils. These pupils originate from 40 counties of Pennsylvania east of Pittsburgh and from the state of Delaware. Owing to the large expense involved in any attempt to visit all the families, the survey had to be restricted to the areas within a few hours' drive from the school. However, the families in 11 counties, including 94 communities of Pennsylvania, were visited, in addition to the families living in 9 communities of Delaware.

Thus the families of 307 children hereafter referred to as the "index cases" were investigated. These, as is seen, represent over half of the resident school population. The survey required nine months, from January to September 1939, and was conducted by a trained social worker.

The forms (A and B) employed for recording the data indicate the scope of the information obtained. Whenever possible the data on form A were verified through contacts with other members of the family, and the data on both forms were also checked with the information given on admission to the school by the parents, guardians or physicians of the children constituting the index cases.

Experience has shown that in a survey covering a large number of families accurate responses cannot be expected with regard to ascendants beyond the third generation or to collateral relatives beyond the second generation. This proved

FAMILY SURVEY—PENNSYLVANIA SCHOOL FOR THE DEAF

(1) (I. C. Pat. Mat.) Family Sheet

A

(2) Name I. C. ..... (3) No. ..... (4) Date of interview ..... (5) Relation between husband and wife.....

8	Name	Birthplace	Date	Age		Present Health or Cause of Death	Hospitalizations Dates Place of Death	Ear Diseases		Auditory Difficulties			
				Living	Dead			Type	Age of Onset	Type	Age of Onset	Duration	Physician or Clinic
		9	10	11	12	13	14	15	16	17	18	19	20
6	Husband:												
	Wife:												
7 Offspring or pregnancies	1.												
	2.												
	3.												
	4.												
	5.												
	6.												
	7.												
	8.												
	9.												
	10.												
	11.												

(21) For I. C. Family: Father's occupation and duration..... Rental estimate ..... per mo.

Employed for ..... years; sporadically unemployed since .....

Unemployed since ..... On relief since .....

Notes:

Degree of cooperation by interviewed: .....

History obtained from ..... by .....

B FAMILY SURVEY—PENNSYLVANIA SCHOOL FOR THE DEAF

I. C. and Sibs history

1. Index Case Name.....
2. Order of Pregnancy ..... Terminated: Misc. SP. ab. In. ab. S. B. L. B.
   Date of termination and duration ..... Name L. B. .....
3. Prenatal care: none P. E. ..... times during pregnancy.
   Name physician or clinic .....
4. Occupation before pregnancy ..... number of years.....
5. Occupation during pregnancy ..... until .....month.
6. Housework in general during pregnancy: none less than usual usual more than usual.
7. Pregnancy hist.; neg. per. vomit edema toxemia eclampsia neph. others.....
8. Termination: at home unattended attended by midwife by nurse by physician at clinic (name).....
9. Termination: spontaneous surgical intervention (specify).....
10. Diseases other than obstetric during pregnancy.....
11. Used aspirin quinine other drugs (specify).....during pregnancy as usual more than usual.
12. If L. B.: Phys. anomaly at birth: none yes (specify).....
13. Breast fed yes until ..... never why?.....
14. Growth, stature: slow average rapid
    Growth, weight: slow average rapid
15. Walked at ..... First words: never talked only mama at .....
    Only a few words at ..... Intelligible at .....
16. Diseases at ages: .....
17. Injuries at ages: .....
18. Earaches without discharge: none yes at ages: .....
    Discharging ears: none yes at ages and duration .....
    Surgical intervention: none yes .....
19. Auditory history: negative impaired First noted at.....
    How: .....
    Treated: ..... By .....

to be true in this case, and indeed truly reliable and complete information with regard to the grandparents and the uncles and aunts of the children representing the index cases was seldom procured.

On the other hand, in the great majority of cases really detailed information relative to the parent generation and the prenatal and neonatal history of all the siblings was obtained. Environmental factors during both these periods were explored in considerable detail, such as the mother's occupation before and during each pregnancy, her health and medical treatment, if any, with particular reference to the use of drugs generally considered as contributory to this type of deafness.

Factors in regard to nutrition and health during infancy and the child's physical development were noted and recorded, special attention being paid to the age at which hearing disability and speech retardation were first noted.

Obviously, the procural of such intimate details required a high degree of experience and tact on the part of the questioner. Attesting to the excellence of this particular part of the effort is the fact that in only 1 case was difficulty encountered, although many of the interviews required as much as two hours for completion. Whenever possible they were made by appointment, and always with a preliminary letter of introduction from the superintendent of the school.

CATEGORIES OF TYPES OF DEAFNESS

The pupils of a school for the deaf may be broadly classified with reference to the origin of their deafness as follows:

1. Children born deaf, as a result of (*a*) syphilis in the mother, (*b*) trauma in utero or at birth or (*c*) some cause other than that in *a* or *b*.

2. Children manifesting deafness after birth.

On the basis of the accumulated knowledge on the subject, it can be presumed that all or the majority of cases grouped under 1 *c* constitute instances of deafness transmitted through inheritance. It is the purpose of the present study to weigh the evidence on this point, to determine whether this type of deafness actually differs from the others with regard to familial concentration of the same or other kinds of deafness and, if so, to determine how the inheritance is probably transmitted.

The term "embryonic deafness" is here applied to the type of deafness indicated under 1 *c*, that is, to congenital deafness not associated with a maternal history of trauma or syphilis at pregnancy. The term "congenital" cannot be properly used since not all cases of congenital deafness are included, nor does one wish to employ such terms as "biologic," which are loosely defined in the textbooks. For the purposes of this report the deafness is classified as either embryonic or nonembryonic. In the latter category are included the cases of congenital deafness due presumably to syphilis or to trauma, as well as of auditory impairment manifest after birth. This dichotomous classification was employed in segregating not only the pupils of the School for the Deaf but their siblings, parents and relatives who have impaired hearing.

In classifying the subjects who became deaf in early life in one of the two categories, the first, and most difficult, step was to determine whether the deafness was congenital. The statement "He was born deaf" may mean nothing more than poor observation on the part of the parent. Therefore, the main criterion employed in the actual grouping of the data was whether or not the child ever used articulated speech. If it was stated that the child was born deaf and yet the history showed that without special training he acquired a vocabulary, the auditory impairment was classed as nonembryonic. On the other hand, if the child never spoke but had in infancy (or at birth) suffered a cranial injury or a severe systemic or otic infection, any of which was regarded by a physician as cause of the auditory defect, then the deafness was also classified as nonembryonic. Of course, as is realized, in the latter category were placed all the auditory impairments which occurred after infancy, regardless of their nature.

By means of the aforespecified criteria, it was possible to classify easily the majority of the cases of deafness among subjects of this inquiry and their relatives. However, in a few instances the information given

in the history, particularly that of the index case child and his or her siblings for whom detailed data were obtainable, was to some degree inconsistent. For example, a child is supposed to have become deaf at 5 years of age, after a head injury due to a fall; yet it is also known that the child never learned to talk. In the preliminary segregation of the data, the deafness in such a case has been labeled "probably embryonic."

As is seen, the foregoing classification, while it takes into account the history of onset of deafness as it may be given by the deaf person or by his relative, is essentially based on the more obvious and objective norm of speech development. Intelligent employment of this norm could reduce the time range of uncertainty regarding the type of deafness studied to the first twelve months of life, or maybe earlier. Therefore, in accordance with the stated objectives of this inquiry, it is of practical importance

TABLE 1.—*Size of Family and Sex Ratio of the Sibships of Children Representing the Index Cases*

	Type of Deafness in Index Case		
	Embryonic	Probably Embryonic	Non-embryonic
Number of index cases	116	8	183
Total number of recorded pregnancies of mothers of children in index cases	587	35	844
Percentage of stillbirths, abortions, miscarriages and neonatal deaths among pregnancies	7.7	2.9	7.8
Live births per family	4.7	4.3	4.3
Percentage of males among live births	52.4	58.8	52.2

to note whether the application of this norm is adequate to separate the deafness secondary to factors which are controllable from the deafness that may have its roots in the biologic constitution of the child.

FREQUENCY OF EMBRYONIC AND NONEMBRYONIC AUDITORY IMPAIRMENT AMONG CHILDREN CONSTITUTING THE INDEX CASES AND THEIR RELATIVES

On the basis of the criteria described, the 307 children representing the index cases were grouped into 116 with embryonic deafness, 183 with nonembryonic deafness and 8 whose deafness was probably embryonic. Disregarding the last group, one can conclude that 35.8 per cent of the population of a school for the deaf was composed of children whose deafness presumably occurred before the first year of life and was not due to injury or disease at birth or afterward.

The data regarding size of family, sex ratio and frequency of stillbirths and neonatal mortality for the three groups of children are summarized in table 1.

It will be noted in table 1 that between the two groups, the children with embryonic and those with nonembryonic deafness, there existed practically no difference relative to the number of live births per mother, the frequency of miscarriages, stillbirths and neonatal deaths and the percentage of males among live births. With reference to the first two variables, the figures are somewhat higher than those observed at present in the general population, but they probably reflect the socioeconomic status of the families concerned and their racial origin. More males were also included among the offspring of the families studied. However, the discrepancy between the observed and the expected values is so slight in the case of the two major groups that its significance is not appreciable with regard to the problem investigated.

TABLE 2.—*Frequency of Deafness Among Siblings, Parents, Uncles and Aunts and Grandparents of Index Case Children* *

Kinship	Type of Deafness in Index Case	Number	Percentage of Relatives with Deafness			
			Embryonic	Probably Embryonic	Non-embryonic	Good Hearing
Siblings.....	Embryonic.....	426	14.3	1.7	4.0	80.0
	Probably embryonic..	26	3.8	19.2	0	77.0
	Nonembryonic.....	595	0.5	0.2	4.2	95.1
Parents.....	Embryonic.....	232	7.3	0	6.9	85.8
	Probably embryonic..	16	18.8	0	18.8	62.4
	Nonembryonic.....	366	0	0	4.6	95.4
Uncles and aunts	Embryonic.....	712	4.1	0.2	2.0	93.7
	Probably embryonic..	46	8.7	2.2	4.3	84.8
	Nonembryonic.....	950	1.1	0.3	0.9	97.7
Grandparents....	Embryonic.....	301	1.7	0	6.3	92.0
	Probably embryonic..	20	0	0	0	100.0
	Nonembryonic.....	416	0	0	3.8	96.2

* Only relatives whose hearing status is known are included.

The prevalence of deafness among the relatives of the three groups of children is shown in table 2. The following main facts emerge from this tabulation:

1. Auditory impairment was more prevalent among the relatives of the children with embryonic deafness than among those of the children with nonembryonic deafness. It seems that the familial concentration with reference to the relatives of the children the onset of whose deafness was probably embryonic resembled more closely that noted for the subjects with embryonic deafness.

2. Between the families of the children with embryonic deafness and those of children with nonembryonic deafness, the difference in prevalence of deafness was less marked when respective groups of uncles and aunts and of grandparents were compared. This is as it should be when one is dealing with familial characteristics and is probably more apparent because the prevalence figures for these kin are influenced greatly by

completeness and accuracy of the reported history, which could not be checked.

3. While the frequency of nonembryonic deafness was slightly higher among the parents, uncles and aunts and grandparents of the children with embryonic deafness than among those of the children with nonembryonic deafness, the real difference between the relatives of the two groups of children was manifest with reference to the incidence of embryonic deafness. Thus, 14 per cent of siblings and 7.3 per cent of the parents of the index case children with embryonic deafness had this type of auditory impairment, as compared with 0.5 and 0 per cent, respectively, of the siblings and parents of the index case children with the nonembryonic type of deafness.

4. The frequency of nonembryonic auditory impairment among the siblings of the index case children with nonembryonic deafness was of the order of that to be expected in any population of children. It should be added that the prevalence of auditory defects in the parents, uncles and aunts and grandparents of the index case children was approximately the same for the two sexes. The figures according to sex were therefore omitted.

Two inferences can be drawn from these observations. First, by determining whether the child learned to articulate at all in infancy, one can classify the deafness observed in a population in a school for the deaf into two types with reference to familial concentration. One type, the embryonic, manifests obviously a familial tendency; the other, the nonembryonic, exhibits no such tendency. The absoluteness of the statement regarding the criteria for segregation must be tempered by the consideration that attention was especially given to the history and evidence of syphilis in the mother or of head injury at birth or immediately thereafter in the child.

If it is accepted that the information obtained for the siblings was more accurate than that obtained for the other kin, it may be inferred also that the presence of embryonic deafness in a family does not apparently render the members of the family more liable to other forms of auditory impairment. To support such a view one can add data on the incidence of otitis media among the siblings of the two groups of children constituting the index cases. Of the brothers and sisters of the index case children with embryonic deafness, a history of otitis media was elicited for 8.7 per cent; among the siblings of the children with nonembryonic deafness the rate equaled 8.1 per cent.

In concluding that the deafness classified here as nonembryonic does not demonstrate a familial tendency an obvious reservation must be made. By the nature of the segregation, cases of this type of deafness represent an extensive mixture of etiologic types of impairment. It could

well be that careful study of a number of cases of a single type might reveal one or more in which a familial tendency was indicated.

PROBABLE MECHANISM OF HEREDITARY TRANSMISSION OF EMBRYONIC DEAFNESS

The existence of a familial tendency to a disease or condition does not per se presuppose that the disease or condition is inherited. However, the accumulated evidence seems to point rather definitely to the inheritance of the form of deafness called embryonic in this paper. Accepting such an assumption, it is of practical interest to know whether the inheritance is of the type that is transmitted according to a stable and simple pattern. On this point the majority opinion is that this form of deafness is transmitted according to the mendelian scheme involving a single recessive gene. In the following brief analysis [5] it is attempted to determine whether the data of this investigation support such a view. It should be noted immediately that among the parents of the index case children there was no instance in which the father and mother were first cousins. This contrasts sharply with the observations reported by others and cannot be easily explained without further analysis, impossible at this time. However, the following factors may have contributed to the absence of consanguinity in the parents of this sample of children: (1) Deafness (all forms taken together) is not a rare condition; (2) the native-born parents originated from a densely populated section of the country, and (3) the foreign-born parents arrived in this country before marriage. Factors 2 and 3 were obvious in this material and with factor 1 tended to increase the probability of exogamous marriages.

When the index cases were arranged according to whether one, none or both of the parents had impaired hearing and the number and proportion of offspring (including the children representing the index cases) with embryonic and nonembryonic deafness and with good hearing were calculated for each group, the results shown in table 3 were obtained.

The results with reference to the cases of nonembryonic deafness may first be considered. In three families of children, both parents had nonembryonic deafness, but, except for the 3 index cases, no instance of nonembryonic deafness occurred among the offspring. Moreover, it will be noted that the frequency of deafness among the offspring when one of the parents was deaf was actually less than that observed when

5. The reader who desires to become acquainted with the logic and technic of the analysis can refer to the excellent articles by Hogben (Genetic Analysis of Familial Traits: I. Single Gene Substitutions, J. Genetics **25**:97-112, 1931; II. Double Gene Substitutions, with Special Reference to Hereditary Dwarfism, ibid. **25**:211-240, 1932).

neither of the parents exhibited auditory impairment. When these observations are considered together, the conclusion reached previously that hereditary transmission is not involved in nonembryonic deafness is further confirmed.

TABLE 3.—*Parental Matings and Frequency of Deafness Among Offspring in Families in Which Index Cases Occurred*

Parental Matings	No. of Matings	Number of Offspring: Type of Deafness: Embryonic	Probably Embryonic	Non-embryonic	Good Hearing	All Offspring
A. Embryonic Deafness						
Good hearing x Good hearing	93	138	6	12	285	441
Good hearing x Nonembryonic deafness	11	17	0	2	44	63
Nonembryonic deafness x Nonembryonic deafness	0	0	0	0	0	0
Embryonic deafness x Nonembryonic deafness	5	8	0	2	5	15
Embryonic deafness x Good hearing	2	2	0	1	7	10
Embryonic deafness x Nonembryonic deafness	5	11	2	0	0	13
B. Nonembryonic Deafness						
Good hearing x Good hearing	169	3	0	192	522	717
Good hearing x Nonembryonic deafness	11	0	1	13	42	56
Nonembryonic deafness x Nonembryonic deafness	3	0	0	3	2	5
Embryonic deafness x Nonembryonic deafness	0	0	0	0	0	0
Embryonic deafness x Good hearing	0	0	0	0	0	0
Embryonic deafness x Embryonic deafness	0	0	0	0	0	0

When attention is turned to the sibships in which an index case of embryonic deafness occurred, several illuminating facts are revealed. Of 13 offspring of parents both of whom had embryonic deafness, 11 had embryonic deafness and the remaining 2 had deafness which was probably of embryonic type. These 2 children were twins of the same sex who had simultaneously suffered an attack of influenza when 16 months of age and thereafter were adjudged by a physician to have become deaf. However, neither of the twins had ever articulated words preceding the

attack of influenza. Were it not for the alleged statement of the physician, the twins would be classified with the embryonic deaf. If they are so classified, then all the offspring of parents each with embryonic deafness had the same type of auditory impairment. Therefore, the observations agree perfectly with the expectation that a single recessive gene is the factor in the inheritance of embryonic deafness. Assuming the transmission of embryonic deafness by a single recessive gene, each of the 93 pairs of parents with good hearing and of the 11 pairs one of whom had nonembryonic deafness must have "carried" the gene for deafness. It can be shown by computations based on the theory of probabilities, the size of the sibship being taken into account, that from these matings one should expect 172.1 ± 8.1 children with embryonic deafness. As is seen in table 3, of parents neither of whom had embryonic deafness, 138 children with embryonic deafness and 6 with deafness which was probably embryonic were born, and of parents one of whom had a nonembryonic type of deafness 17 offspring with embryonic deafness were born. Thus, a total of 161, instead of 172, deaf children, were born to the 104 pairs of parents. However, the difference between the actual and the calculated figure is small enough to be due to chance, and so further support is given to the view that the embryonic type of deafness is transmitted through a single recessive gene. The consistency of theory with observation is evident again when the expected number of deaf children is calculated for the offspring of parents only one of whom had embryonic deafness. From the 3 matings in which one parent had good hearing and the other embryonic deafness and the 4 matings in which one parent had nonembryonic and the other embryonic deafness, 10 offspring with embryonic deafness were produced. On the basis of inheritance through a single recessive factor, the expected value is 13.8 ± 2.2, and here, again, the difference between the observed and the calculated figure is not statistically significant.

These observations, therefore, are in sufficient agreement with the opinion that the type of deafness called embryonic in this study is transmitted from parent to offspring by a single recessive factor, or gene. As is known, this would mean that (*a*) of the marriage involving a man and woman both of whom have embryonic deafness, all the offspring will be deaf; (*b*) of the marriage involving only one partner with embryonic deafness, either one-half the children or none at all will have embryonic deafness, depending on whether the parent with good hearing is a carrier of or is completely free from the gene in question, and (*c*) of the marriage involving one partner who has good hearing but is a "carrier" of the gene, either 25 per cent or none of the offspring will have embryonic deafness, depending on whether the other partner with good hearing is also a carrier.

SUMMARY

The results of this analysis reveal, first, that from the history of the onset and immediate cause of deafness it is possible to differentiate among the pupils of a school for the deaf the children whose deafness is probably inherited and those whose deafness is acquired. The former apparently constitute about 35 per cent of the school population. It is interesting to point out that Lenz,[6] from a consideration of deaf mutism in Germany, concluded that in 25 to 30 per cent of cases the deafness was inherited. From the analysis made in the present paper it seems that the deafness is transmitted according to the mendelian pattern involving a single recessive gene.

The significance of these observations lies in their contribution to a clarification of the problem of preventing deafness of the type investigated. On the assumption that the inference regarding heredity is entirely correct, it is immediately perceived that, whether or not a history of deafness in the immediate family can be elicited, a person with embryonic deafness will transmit the particular gene to his offspring and the chances that the transmission is manifest in the offspring will depend on the hereditary makeup of the spouse. It will be further realized that the presence of embryonic deafness among the brothers and sisters, even though the subject has good hearing or the acquired type of deafness, is sufficient to arouse suspicion that the person is a carrier of the recessive gene for deafness.

If the figures on the prevalence of nonembryonic deafness in the school surveyed are applicable to all schools for the deaf, it appears that in 65 per cent of the population of such schools factors in the environment are in the main responsible for the deafness. The diseases and conditions that may be included in the factors are those related to the uterine environment and have yet to be adequately evaluated. Data bearing on this point will be presented in a subsequent article, in which a further analysis of the information resulting from the family survey will be reported.

6. Lenz, F.: Die krankhaften Erbanlagen, in Baur, E.; Fisher, E., and Lenz, F.: Menschliche Erblichkeitslehre, Munich, J. F. Lehmanns Verlag, 1927.

ESTROGENIC IMPLANTS IN THE TREATMENT OF ATROPHIC RHINITIS

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LOS ANGELES

Until recent years atrophic rhinitis, or ozena, has been most resistant to therapy. However, the work of Mortimer and associates,[1] Blaisdell [2] and Dorochenko [3] has opened up a new type of treatment, based on the theory or assumption that atrophic rhinitis, or ozena, may be associated with an endocrine deficiency. The existence of a nasogenital relationship has long been known. Consequently, it is not surprising that estrogen and androgens should be tried in the treatment of nasal disease. One could reason, in view of known nasogenital relationships, that if estrogens are helpful in the treatment of atrophic vaginitis, favorable results might be obtained, as well, by estrogenic treatment of atrophic rhinitis.[4]

Mortimer and his associates [1] treated 31 female and 7 male patients with an estrogen by nasal insufflation, with varying degrees of success in 81 per cent of his group. Blaisdell [2] similarly sprayed estrogenic substances in sesame oil into the noses of more than 60 persons and found that the larger number of these patients were helped in varying degrees. Dorochenko [3] also used local applications. His product was a specially prepared, sterilized extract of urine from pregnant women. Nasal tamponade was discontinued after trial in a small group, because of the disagreeable odor. However, when he injected the endocrine extract intramuscularly in a series of 38 cases, he found an amelioration of the disease in all but 2 after eight to ten injections. Twenty to thirty injections were usually spaced over a period of one to two months, depending on the response of the patient. In most of the cases the

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1. Mortimer, H.; Wright, R. P., and Collip, J. B.: Atrophic Rhinitis: The Constitutional Factor and the Treatment with Oestrogenic Hormones, Canad. M.A.J. **37**:445 (Nov.) 1937.

2. Blaisdell, I. H.: The Use of Estrogenic Substances in Atrophic Rhinitis, Laryngoscope **48**:699 (Oct.) 1938.

3. Dorochenko, I. T.: Un nouveau principe de la thérapeutique de l'ozène, Acta oto-laryng. **25**:542, 1937.

4. Since Dorochenko, Blaisdell and Mortimer and his co-workers apparently did independent similar work on the estrogenic treatment of ozena, it is difficult to determine priority in the discovery of this mode of treatment among these investigators. Two papers were published late in 1937 and one late in 1938.

condition was severe and of long standing, but after the treatment the patients experienced increased nasal secretion, and objectively nasal hyperemia and mucosal infiltration were noted. Fetid odors also disappeared.

Although topical estrogenic treatment of ozena has now apparently become well established, it offers certain drawbacks. It requires much and frequent local attention. Consequently, when in the fall of 1938 I became aware of the work of Deanesly and Parkes,[5] it occurred to me that if the work of previous investigators [6] could be combined, a new avenue for the treatment of atrophic rhinitis might be opened.

Briefly, the work of Deanesly and Parkes consisted of demonstrating the success of implanting pellets of estrogenic and other endocrine substances subcutaneously in animals. Definite therapeutic effects were observed, and, what is more important, potent effects were noted over a long period. These workers removed some of the implanted pellets from time to time and weighed them, thus determining the rate of absorption. Estrone (theelin) and estradiol (dihydrotheelin) were found to be absorbed at the rate of 2.5 to 9 per cent per month. Testosterone and progesterone were absorbed much more rapidly.

Hamilton and Dorfman [7] studied the influence of the vehicle on the duration and intensity of the action of androgens and found that pellets of testosterone propionate gave the longest and most pronounced stimulation in comparison with a number of other injection vehicles, including oil. Thorn [8] and his associates found that pellets of desoxycorticosterone acetate exerted potent therapeutic effects when implanted in adrenalectomized dogs. Removal of the pellets was followed by disappearance of these effects. These authors have also obtained favorable results in 4 patients with Addison's disease. Vest and Howard [9] have used implants of a crystalline androgenic substance with success in cases of hypogonadism.

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5. Deanesly, R., and Parkes, A. S.: Further Experiments on the Administration of Hormones by the Subcutaneous Implantation of Tablets, Lancet **2**:606 (Sept. 10) 1938.

6. Mortimer, Wright and Collip.[1] Blaisdell.[2] Dorochenko.[3] Deanesly and Parkes.[5]

7. Hamilton, J. B., and Dorfman, R. I.: Influence of the Vehicle upon the Length and Strength of the Action of Male Hormone Substance, Testosterone Propionate, Endocrinology **24**:711 (May) 1939.

8. Thorn, G. W.; Engel, L. L., and Eisenberg, H.: Treatment of Adrenal Insufficiency by Means of Subcutaneous Implants of Pellets of Desoxycorticosterone Acetate (A Synthetic Adrenal Cortex Hormone), Bull. Johns Hopkins Hosp. **64**: 155 (March) 1939.

9. Vest, S. A., and Howard, J. E.: Clinical Experiments with Androgens: A Method of Implantation of Crystalline Testosterone, J. A. M. A. **113**:1869 (Nov. 18) 1939.

NATURE AND SOURCE OF MATERIAL

As a basis for this study, 24 patients from the White Memorial Clinic were treated. Twenty were women, and 4 were men. All had atrophic rhinitis, or ozena, in various stages and of varied duration. Some possibly had chronic purulent sinusitis as a complicating factor, and indeed in some cases it was difficult to determine whether atrophy, crusting and other manifestations represented true ozena or were merely a result of severe long-standing purulent sinusitis. Implants were made only in cases which could reasonably be classified as cases of ozena. Certain patients with apparently typical ozena show marked roentgenographic evidences of maxillary sinusitis, yet puncture and irrigation of the sinus yield no pus. Indeed, in many cases of atrophic rhinitis roentgenographic views of the paranasal sinuses reveal some clouding.

The compressed pellets [10] were a little smaller than ordinary hypodermic tablets, and each consisted of 20 mg. (240,000 rat units) of pure crystalline alpha estradiol. Though the pellets were sterile and were encased in individual sterile sealed glass ampules for double safety, each tablet was removed from its ampule and individually boiled in water for five minutes just before use. Only 2 or 3 out of 30 disintegrated during this process.

TECHNIC OF IMPLANTATION

The subcutaneous tissue of the abdomen was selected as the site for implantation of the estrogenic pellets. Since it was not known what local reactions might take place, it was decided not to use the right side of the abdomen, because of possible confusion with the common causes of intra-abdominal pain on that side. Instead, the left upper abdominal quadrant was chosen. In 1 case the right upper quadrant was used, however.

After antiseptic treatment of the skin, the selected site was anesthetized by injection of a small amount of 1 per cent procaine hydrochloride. A 1 cm. incision was made down to the subcutaneous fat, and a pocket was made in the fatty tissue by blunt dissection. The sterile pellet was then introduced into the pocket, to a depth of 10 to 15 mm., by means of a forceps. The edges of the wound were approximated by skin clips. No pain or other local reaction was noted except the minor incisional trauma, and no infections occurred.

COMMENT

There is no special reason to believe that the site chosen for implanting is better than other sites. Other authors [9] have used sites such as

10. The pellets of alpha estradiol were supplied by Roche-Organon, Inc., Nutley, N. J.

the legs, arms and back. Likewise, the technic described for implanting the pellets represents only one method. An interesting and promising injector has been devised for this type of work.[9] I have had no experience with this injector.

PLAN FOR TREATMENT

In order to make the observations as accurate as possible, it was planned to start no new treatment concurrently with the implantation of the crystalline estrogen. My associates and I did our best to hold to this principle, though we naturally had no control over treatments administered to the patients for other ailments by other physicians. If a patient had been in the habit of cleansing the nose daily by irrigation, he or she was advised to continue as before. Otherwise, irrigation was not resorted to. Most patients, however, had irrigated their noses previously. Individual exceptions to these principles will be noted in the respective case reports.

At the time of the infrequent (one, two or four month) visits to the clinic no treatment was carried out except the occasional removal of crusts with a cleansing spray and irrigation. This aided in viewing the mucosal changes. It is unlikely that these procedures affected the general course of the disease or that the outcome of the estrogenic treatment was influenced thereby. In 3 cases a preparation of estrone in oil had been previously employed without giving adequate relief. In 1 of these there was a waiting period of two months before implanting, and in the other 2 cases, a period of six months.

When individual patients appeared to have reached maximum improvement or had failed to respond to treatment, supplementary therapy was sometimes ordered. The results obtained after the supplementary therapy was instituted are not included in the analysis, since the cases were removed from the series at the time the supplementary treatment was begun. Only results obtained up to that time are used in drawing conclusions.

CASE REPORTS

The 24 cases reported here provide specific examples of the common clinical picture, the dosage and the effects observed in typical patients:

CASE 1.—A Mexican woman aged 30 complained of having had offensive breath since the age of 10. This had been severe for two or three years. Nasal obstruction had been present for two years. The sense of smell was impaired, but taste was normal. For the previous one or two years greenish crusts had been present, which were hard to dislodge. One month following implantation of 1 pellet (20 mg.) of alpha-estradiol, she reported improvement, with less dryness and discharge.

CASE 2.—A white woman aged 61 complained of nasal dryness and crusting of thirty years' duration, which had been worse lately. There was evidence of

chronic maxillary sinusitis, with mucosal thickening, demonstrable by roentgen rays. During the ten month period following the implantation of the estrogen she was seen five times. At every visit she reported much improvement, with less crusting and dryness. The nasal mucosa became more normal in appearance.

CASE 3.—A Mexican woman aged 28 stated that at the age of 22 she began to have headaches and vertigo. Severe foul nasal discharge and crusting also began at that time. The sense of smell had decreased. The menstrual history was essentially normal. Examination showed much nasal crusting and atrophy, chiefly on the left side. The presence of a mild pansinusitis was suggested by roentgen examination. Although the clinical diagnosis of ozena was questioned, biopsy of the turbinates showed typical histologic changes. The breasts were sore for ten days after implantation of the estrogen. Investigation after three months of treatment revealed less crusting but more fluid discharge and more headache. The odor was also said to be worse.

CASE 4.—A white woman aged 55 complained of having had dryness of the nose since the age of 10. The menarche occurred at 13, and a bilateral oophorectomy was done at the age of 38, but no menopausal symptoms had been noted. For two months after implantation of the estrogen her breasts were very tender. Her nasal trouble abated rapidly, and when the patient was last seen, nine months after implantation, she reported much improvement, with less crusting. Some crusts were still visible, however, but there was no appreciable odor.

CASE 5.—A Negro woman aged 25 complained of an odor from the nose for two years, with anosmia as well as nasal dryness and much crusting. Roentgenograms showed evidence of pansinusitis. Two months after implantation of the estrogen there was less headache, less soreness of the nose, less "sniffling," softer crusts and only occasional odor. The mucosa appeared red and moist. At the end of two years the patient's condition is still definitely improved, though there is some soft crusting and foul odor. (At her last visit she revealed that she had begun to wash her nose at the same time that the implantation was done. We had not known of this fact before. She expressed the belief, however, that the irrigation is not the only source of improvement, since now she finds it necessary to wash her nose only very infrequently, as compared with the daily washings two years ago. Now she sometimes allows an interval of two weeks between irrigations.)

CASE 6.—A Mexican woman aged 23 complained of crusting in her nose for one year, with headaches. Atrophy was evident. She had received some relief from spraying 1 oz. of amniotin in oil into the nostrils but had discontinued this six months previously. Almost two months after the implantation of the estrogen she reported partial improvement. The headaches had disappeared, and the crusts appeared less dry. After five months, though her condition was improved, the odor had increased, as had the fluid discharge. The site of the implantation was reexplored, but the old pellet was not found. A new one was inserted, and four months later the patient felt improved. The odor was less, and there were fewer crusts. The nasal mucous membrane appeared moist. The total observation time was nine months.

CASE 7.—A Mexican woman aged 23 complained of headache (frontal and orbital) and of mucopurulent nasal discharge of three years' duration. Roentgenograms showed pansinusitis. The nose was filled with a green cheesy discharge. Biopsy revealed changes typical of atrophic rhinitis. Two months after implantation of the estrogen the headaches were less. Crusts still formed but were easier

to dislodge. The nasal mucosa appeared quite normal. She continued to improve, and the crusts disappeared, but after five months headaches began to reappear. After about six months a second pellet was implanted. Four months later still very little crusting was noted, but mucopus was abundant. The mucosa was fairly normal in gross appearance. The total observation time was eleven months.

CASE 8.—A Mexican woman aged 19 complained of nasal odor and discharge of seven years' duration. The intranasal appearance was typical of ozena. Four months after implantation of the estrogen no improvement was noted. The use of amniotin as a nasal spray was ordered. The patient has not returned.

CASE 9.—A Mexican woman aged 49 had had intranasal operations on the maxillary sinuses at the age of 33. She had washed her nose daily to remove crusts. Crusts and atrophy were noticeable. Roentgenograms showed pansinusitis. She had used amniotin in oil as a spray, obtaining some improvement, which, however, was not maintained. Two months after she discontinued the use of the estrone spray an implantation of estrogen was made. Improvement was noted in one month. After two months she reported a few pains in her breasts. When last seen, more than twenty-three months after the implantation, her condition was still improved, with less headache, crusting and dryness. Inasmuch as she did not have anosmia, she was also able to report the disappearance of the foul nasal odor.

CASE 10.—A white woman aged 22 complained of nasal crusting of four to five years' duration. A frontal headache had been present for one year. She had received some help from the use of amniotin spray at home for eight months. Roentgenograms showed slight clouding of the maxillary sinus. Six months after she last used the estrone spray an implantation of estrogen was made. She improved only in that crusts were less dry and easier to dislodge. When last seen, after ten months, she reported slight improvement only.

CASE 11.—A Mexican woman aged 27 complained of low frontal headache and nasal and postnasal discharge of several years' duration. At the age of 23, after having had ether anesthesia for childbirth, she first noted anosmia. She said that her sister had the same difficulty. Examination showed much nasal crusting and mucopus. Culture of the crusts revealed the presence of an Aspergillus. When seen three months after implantation of the estrogen she reported less crusting, more fluid discharge and less soreness in the nose. The anterior part of the nasal mucosa appeared moist, but there was much crusting posteriorly. The aspergillus infection was not treated, because the patient failed to return to the clinic as requested.

After twenty-two months she finally returned and reported that the condition had been improved for six months following the implantation. Then her old symptoms gradually began to return, though she still feels that her condition is slightly improved. A reimplantation has been advised. A new culture for fungi has been made. However, the mold has not been fully identified as yet.

CASE 12.—A white woman aged 40 complained of much nasal dryness and crusting since childhood. The large crusts were dislodged with much difficulty by means of nasal douches. Some of the crusts were reported to have been at least 2 inches (5 cm.) long, green, and hollow. Seven years previously a diagnosis of atrophic rhinitis had been made in the White Memorial Clinic. Local treatment prescribed then had apparently helped her but little.

Examination revealed crusting in both nasal fossae (worse on the left side) and in the nasopharynx. There was relatively little turbinal atrophy. Two months

after implantation of the estrogen she noted less crusting, less dryness and less odor. At each following visit she reported improvement. After sixteen months she had a severe attack of influenza, which caused a partial relapse in her nasal trouble, though after twenty-two months she still felt better than she did before treatment was instituted. In view of the partial relapse, a reimplantation is contemplated.

Prior to the implantation she found it necessary to irrigate her nose as much as three times daily. Now she irrigates it only once in several days. Previously her menstrual periods occurred every two or three weeks and the flow was heavy. After the treatment the menses became more regular and the flow less. Now the flow is very slight and occurs at intervals of six weeks.

CASE 13.—A Mexican aged 33 complained of nasal obstruction of a year's duration, with dryness in the nose and frontal headache. Crusting had been present since the age of 12. He could note some foul odor himself. Some one in the clinic advised a submucous resection, and this was done. Five months later symptoms were still present, with much atrophy of the turbinates. An estrogenic pellet was implanted, following which procedure the patient was observed for eight months. He reported much improvement and less crusting and discharge. Headaches disappeared, there was less odor and the nose appeared more moist. About eight months after the implantation the crusts began to reappear, and since no more pellets were on hand, local treatment with tampons was begun.

CASE 14.—A white woman aged 39 complained of nasal catarrh which had been present since the age of 12 and of severe nasal crusting since the age of 31. She returned one month after implantation of the estrogen but had had a severe nasal and chest cold for two weeks, so no accurate observation could be made at that time. At twenty months, however, she returned and reported that she had noted marked improvement for sixteen months. She had had less crusting and discharge, the crusts dislodged more easily and the odor disappeared altogether. Now all her old symptoms have returned. A reimplantation has been advised.

CASE 15.—A white woman aged 65 complained of excessive nasal secretion, nasal obstruction, foul breath and green crusts in her nose since the age of 15. The sense of smell was poor. Much nasal atrophy and crusting were seen. The odor was foul. The condition was believed to be typical ozena. She was observed for five months after implantation of the estrogen. Within one week after the implantation the crusts began to loosen easily and she noted less odor, less crusting and less discharge. When last seen she had no crusts but recently had had a slight increase in mucopurulent discharge. She was then permitted to begin nasal irrigations, which she had never used before.

CASE 16.—A Mexican aged 20 claimed to have had nasal discharge all his life. Roentgenograms showed pansinusitis. One of my associates thought this case to be one of ozena and implanted an estrogenic pellet. After three months there was no improvement, and the use of an oily estrogenic nasal spray was prescribed. Two months later the patient returned, complaining of pain, tenderness and swelling of the mammary glands. There had been some watery mammary secretion. With the region under local anesthesia the pellet was removed. It appeared to be of the same size as the original pellet. Swelling of the breasts had apparently begun after the implantation and had become aggravated after use of the nasal spray. Two weeks after the estrogenic pellet had been removed, the swelling of the breasts had essentially disappeared. Nineteen months after implantation the patient

reported only slightly less crusting and discharge than he had originally. The nasal mucosa was fairly normal and moist. There were no cysts or masses in the mammary glands.

CASE 17.—A Mexican aged 31 complained of nasal discharge and crusting of one year's duration. Many crusts and much mucopus were seen in the nose. Two months after implantation of the estrogen he reported less discharge, less crusting and fewer headaches. Seven months after the implantation he felt very little improved, and other local treatments were begun.

CASE 18.—A Mexican woman aged 23 complained of having had a nasal discharge since the age of 14 and of dryness of one year's duration, with crusting and a foul nasal odor. Four months after implantation of the estrogen she returned to report less drying and crusting in the nose. This improvement began three weeks after the implantation. The odor was worse, however. (The patient in case 19 reported that when she last saw this patient, a personal friend, seventeen months after the implantation, she was still improving. She is now in Mexico.)

CASE 19.—A Mexican woman aged 28 complained of having had postnasal drip and headaches for five years. Much green crusting was noted in the nasopharynx only. The nasal chambers were very roomy, but the case was not considered a strictly typical case of atrophic rhinitis. One month after implantation of the estrogen the patient reported less crusting and dryness. Inspection showed only minor nasopharyngeal crusting. Eighteen months later she felt that she was still improving. She rarely noted crusts, had less discharge, which was more clear, less headache and less odor. The last examination revealed only minor nasopharyngeal crusting. She refused a tonsil and adenoid operation, which was advised.

CASE 20.—A Mexican aged 25 had been treated for syphilis for one year, and the blood still gave a positive reaction on test. Much crusting was seen in the nose, and a foul odor was noted. One month after implantation of the estrogen he reported less nasal discharge but no decrease in crusts.

CASE 21.—A white woman aged 39 complained of noting "scabs" in her nose since her menarche at the age of 13. Her sense of smell had been lost, but her family and friends told her of a disagreeable nasal odor. She was observed for sixteen months after the implantation of the estrogen. The improvement which began in one month still persists, and there is no nasal odor. The bilateral crusts have disappeared, and only occasionally does she wash out a small crust from the left side. Formerly the crusts were three times larger and more frequent. Examination revealed a 5 mm. crust posteriorly on the left side of the septum. The rest of the nasal mucosa appeared quite normal and moist. There is no longer any nasopharyngeal crusting, and the nasopharynx is less dry.

CASE 22.—A white woman aged 31 complained of nasal crusting and a yellow discharge of five years' duration, which had been worse for the past six months. She was observed for three months after the implantation of the estrogen, but no improvement or aggravation resulted. She was removed from the series and other treatment instituted. She menstruated twice in one month after the implantation. (Almost one year after the patient's last visit her father, who also has ozena, returned to the clinic to report that his daughter's nose was "well." This result is not included in the conclusions.)

CASE 23.—A white woman aged 19 complained of a foul odor from the nose of ten years' duration. She also had anosmia. Examination showed extensive nasal crusting and much mucosal atrophy. One month after implantation of the estrogen

she reported slight improvement. At three months she stated that the crusts were not so big or so dry. Her family says the odor is less foul. Examination showed minor crusting high in the nose. At seven months the condition was essentially the same.

CASE 24.—A Mexican girl aged 18 complained of "frequent boils" in the nose and odor from the nose. Examination revealed much crusting. Two months after implantation of the estrogen she began to note improvement. There was less discharge and crusting. She had not used saline nasal irrigations and was shown how to use them. (She continued to improve greatly, but because the adjunct treatment was instituted, we have classified her as only mildly improved. She was last seen fifteen months after the implantation.) She stated that after the implantation her menses were characterized by less pain and flow.

RESULTS

In summarizing the results it seems best to divide the series according to sex. Of the 20 female patients, 13 were definitely improved. Four experienced only mild improvement or were observed for such a short time that improvement cannot be classified as proved beyond doubt. In 3 patients the condition was not improved or was aggravated.

Of the 17 patients who showed some degree of improvement, 10 were observed for periods ranging from nine to twenty-four months. Initial improvement was usually noted one to eight weeks after implantation. In cases 6 and 7 reimplantation was required after four and six months respectively. Whether the periods noted can be regarded as the absorption time for an entire estrogenic pellet is not clear. In cases 5, 9, 11, 12, 14 and 21 reimplantation is contemplated, since the effects of the original pellets implanted sixteen to twenty-four months previously seem to have diminished or disappeared.

In cases 12, 22 and 24 the patients experienced temporary changes in their menstrual periods, the exact significance of which is not clear in such a small series. Two female patients noted temporary tenderness of the breasts.

One of the 4 male patients displayed marked improvement which lasted for eight months. Another experienced mild temporary improvement. The other 2 were not helped. One of the latter noted enlargement and pain in the breasts. The pellet was removed after five months and was found to be apparently the same size as in the beginning. The breast changes seem to indicate that powerful pharmacologic action can be expected from the implanted estrogens.

Generally speaking, the improved patients noted less dryness and crusting, sometimes more fluidity of secretions, sometimes less secretion, often fewer headaches and a general feeling of improvement. At least 2 patients who had experienced trouble in gaining weight noted increases in their weight. Some who had had the disease for many years felt subjectively well. Objectively, less crusting was noted, and the nasal

mucosa was observed to have changed from a dull, dry stage to a more moist and glistening appearance. Clinical improvement from one implantation seems to last from four months to almost two years.

In this series I tried to use only the single treatment, namely, implantation, unless the patient had already used irrigation or other therapeutic aids. However, except in a carefully controlled research series, nasal irrigation and even the use of local nasal estrogenic applications might be advisable as adjunct treatment.

Fifty-four per cent of the patients in this series were Mexicans. In the White Memorial Clinic as a whole, Mexican patients constitute less than 10 per cent of new admissions. Whether this difference indicates the influence of racial, economic or social factors is not clear.

SUMMARY

The treatment of atrophic rhinitis by the subcutaneous implantation of estrogens is here reported for the first time to my knowledge.

Implantation of large unit doses of estrogenic substances exerts a beneficial effect in atrophic rhinitis. Only the female patients in this series seemed greatly benefited.

The exact mode of action of the estrogenic substance is not yet proved.

Further studies by various investigators are indicated.[11]

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11. Since preparing this report I have continued to use the treatment in additional cases, with encouraging results. I have also begun the oral use of stilbestrol in atrophic rhinitis but have no report to make as yet. I suggest other investigators also consider the use of stilbestrol in ozena.

HYPODYNAMIA LARYNGIS

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A well known pathologist recently remarked to me that he dreaded the ordeal of performing an autopsy on newborn infants because so frequently no information is revealed to explain the cause of death. Every hospital staff knows the frequency with which the combination of circumstances—death from respiratory failure and negative autopsy findings—occurs.

A large number of contributions to the literature of recent date have forced a revision, if not a complete rejection, of the previously held concept of the status and the function of the fetal lung. In the past, the lung was looked on as a mass of collapsed inert tissue, waiting for the arrival of birth to set off the spark of its functional activity. Enough has been learned to date to show that, on the contrary, (1) respiratory movements take place in utero at least during the last half of fetal life; (2) that there is a tidal flow of amniotic fluid into and out of the bronchial tree with these movements; (3) that the lung, instead of being collapsed, contains a certain amount of the amniotic fluid and that this fluid is probably essential as a medium for the development of the respiratory epithelium; (4) that as a result, the respiratory and, especially, the bronchial epithelium are subject to the trauma of foreign substances which may be present in the fluid.

Windle and collaborators [1] observed such movements in 3 fetuses, measuring 70 mm., 85 mm. and 85 mm., respectively; in each of these cases, the mother's last menstrual period was reported as occurring not more than eighty-four days previously. Bancroft and Barron [2] from their studies on sheep embryo, concluded that at forty days, generalized mass rhythmic movements occur, not induced by asphyxia. At fifty days, respiratory movements are almost constant, evidently consequent on trivial muscular movements. At sixty days, they are difficult to elicit but can be induced by asphyxia. About the same time, Snyder and Rosenfeld [3] injected india ink into the amniotic fluid of the fetuses of

1. Windle, W. F.; Dragstedt, C. A.; Murray, D. E., and Green, R. R.: A Note on the Respiration-Like Movements of the Human Fetus, Surg., Gynec. & Obst. **66**:987 (June) 1938.

2. Bancroft, J., and Barron, D. H.: Movements in Mid-Fetal Life in the Sheep Embryo, J. Physiol. **91**:329 (Dec. 14) 1937.

3. Snyder, F. F., and Rosenfeld, M.: Intra-Uterine Respiratory Movements of the Human Fetus, J. A. M. A. **108**:1946 (June 5) 1937.

rabbits and recovered it from the lungs of those in which respiratory movements were observed.

Snyder and Rosenfeld [4] also were able to make moving picture records of respiratory movements, and in a recent study, Bonar, Blumenfeld and Fenning [5] made similar records with greater accuracy, besides confirming observations made by Ahfeld [6] and Reiffersheid [7] in 1890 and 1911, respectively.

The sum of these observations leads one to believe that the development and the activity of the lung in utero parallel that of other organs of the body, including the heart, the kidney and the intestinal tract, each of which shows evidence of an increase in its typical activity beginning with the first weeks of fetal life. It is therefore safe to assume that some mucus is prepared by the mucous glands in utero and that this together with the amniotic fluid keeps the surfaces of the alveolar epithelium separated during the course of development and is carried out of the lung by the tidal flow of the amniotic fluid. Considering the presence in this fluid of substances such as bile, meconium, urine, vernix caseosa (including lanugo hairs) and other debris, the production of mucus as a result of this stimulus may be considerable in amount. It is not unreasonable to assume that this foreign matter may be an important contributing cause or even the only cause of inflammatory processes occurring in the fetal lung which were previously attributed (frequently without proof) to congenital syphilis. These findings explain the large quantities of mucus frequently found in the air passages of newborn infants.

In their recent book, "The Larynx and Its Diseases," Jackson and Jackson [8] described under the term "laryngomalacia" a laryngeal condition frequently observed in young children. While the component parts of the larynx cannot be said to be abnormal, the epiglottis is seen to be soft and flabby, pale in color and yielding in position to the slightest external physical influence. The vocal cords are dull, lax and lifeless in appearance. They are blown outward during expiration and fall back

4. Snyder, F. F., and Rosenfeld, M.: The Relation of Intrauterine Respiration to the Development of the Fetal Lung. Anat. Rec. (supp. 3) **67**:47 (March 25) 1937.

5. (*a*) Bonar, B. E.; Blumenfeld, C. M., and Fenning, C.: Studies of Fetal Respiratory Movements: Historical and Present Day Observations, Am. J. Dis. Child. **55**:1 (Jan.) 1938. (*b*) Bonar, B. E., and Fenning, C.: Studies of Fetal Respiratory Movements: Recording Methods, ibid. **55**:322 (Feb.) 1938.

6. Ahlfeld, F.: Die intrauterine Tätigkeit der Thorax-und Zwerchfellmuskulatus, intrauterine Atmung, Monatschr. f. Geburtsh. u. Gynäk **21**:143, 1905; cited by Bonar, Blumenfeld and Fenning.[5a]

7. Reifferscheid, K.: Ueber intrauterine Atembewegungen des Foetus, Deutsche med. Wchnschr. **37**:877, 1911; cited by Bonar, Blumenfeld and Fenning.[5a]

8. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937.

into the rima glottidis on inspiration. While this description covers the anatomic findings clinically observed, it is the opinion of the author that the most important element in the resultant clinical picture is not covered by a purely anatomic descriptive term.

Every one is familiar with the fact that in the first months of life, the muscles of the neck have not developed sufficient power or coordination of their movements to control the position of the head. The anatomic structure, mechanically speaking, is complete and perfect, but the newborn infant must acquire through experience and exercise, first, increased strength of the skeletal muscles and, second, a coordination of the activity of these muscles capable of a satisfactory response to the voluntary or automatic stimuli from the higher centers. The same combination of weakness and lack of coordination is observed in the musculature of other parts of the body, including the larynx.

Drawing of hypodynamia laryngis. In the expiratory phase the larynx has the following characteristics: (1) a dusky red color not deep enough to be described as cyanosis; (2) a flabby epiglottis (not under muscular control); (3) the large wings of the omega-shaped epiglottis thrown outward by the expiratory blast (for the sake of clearness, mucus, which is generally present in quantity, is not shown); (4) lax and bluish pink vocal cords.

It is easy to observe in newborn infants up to the age of 8 to 10 weeks the product of the combined circumstances just described. During expiration, the mucus present in the bronchial tree is forced outward into the hypopharynx. During inspiration this mucus is sucked back into the lung. When the mucus is thick and sticky and the larynx is soft and weak, the return flow carries with it the yielding structures of the laryngeal vestibule. The result is that the aryepiglottic folds, the arytenoid bodies, the vocal cords and even the epiglottis itself cave in and produce complete obstruction. In my opinion, the sequence of the involvement of these structures is in the order already named, i. e., the

aryepiglottic folds, the vocal cords and the epiglottis. It appears, therefore, that the weakness of the intrinsic muscles of the larynx is a more important factor in the production of the collapse of these structures than is their own flabbiness. I believe, therefore, that to this condition should be applied the term "hypodynamia laryngis" or "weak larynx."

Unfortunately, large numbers of patients with this condition are not seen at birth, for the simple reason that an accurate search for the cause of dyspnea in newborn infants is not generally made.

The conclusions drawn before were arrived at after witnessing a number of autopsies of newborn infants without any apparent cause of death and after laryngeal examination of 18 children in the second to fourth months of life who may have escaped death at birth by a narrow margin and suffered continually from dyspnea thereafter or who after a normal birth were thrown into acute dyspnea by a minor respiratory disturbance.

Characteristic in these cases is the disappearance of dyspnea during direct laryngeal examination. The tip of the laryngoscope opens the laryngeal vestibule and breathing becomes quiet and easy unless there is a large amount of mucus. Removal of the mucus, if present, gives complete relief of symptoms so long as the laryngoscope remains in place, but as soon as the laryngoscope is removed, symptoms gradually recur and become worse as mucus reaccumulates. Patients with this condition, although they keep the members of the immediate family in a state of tension and fear, especially at night, rarely, if ever, die from this cause alone. At birth, however, the application of artificial respiration in the presence of this condition forces air out of the lungs, while ingress of air is prevented by the caving in of the larynx already described. Under such circumstances, it is conceivable that artificial respiration, as usually applied, may increase rather than alleviate the dyspnea.

The following case reports are illustrative of the clinical conditions encountered. In all cases, the laboratory findings showed nothing peculiar to or typical of the condition.

REPORT OF CASES

CASE 1.—J. L., a 7 week old white boy, was admitted to the hospital on May 6, 1940 and discharged on May 7. He was a full term infant and had been born by normal delivery. Since birth, the baby's breathing had been stertorous, sometimes amounting to dyspnea. The intake of air was accompanied by a rattling or crowing sound. The symptoms were usually worse during the day and particularly when the baby was active. Sleep had been broken to some extent by this complaint, but he had never suffered from fever or acute illness. During the three days prior to admission to the hospital, the mother had been alarmed by an increase in crowing sounds and apparent difficulty in breathing.

Physical examination revealed a normal infant of stated age asleep in bed without any evidence of difficulty in breathing. As soon as the baby was aroused,

however, a rattling sound developed with inspiration. This quickly became crowing in nature and finally, when the child cried, could be described only as inspiratory obstructive dyspnea. There was no difficulty on expiration, and strong pure vocal tones were produced. It was remarkable that in spite of difficulty in breathing, no cyanosis appeared.

Direct laryngoscopy was done on May 7 with a newborn infant's laryngoscope. The hypopharynx was examined. Considerable quantities of thick mucus were seen flowing in and out of the larynx with the respiratory movements of air. Much of this was aspirated but quickly reformed. The epiglottis was of the typical omega shape. The mucous membrane was so thick that the internal surfaces of the epiglottis almost touched each other. Apart from dulness in color and flabbiness in tone of the vocal cords, the larynx was normal. This infant has been seen at regular intervals since this time and has developed normally.

Case 2.—S. G., a 4 month old white boy, was admitted to the hospital on Nov. 10, 1938, and discharged November 30. The infant was normal in all respects until six hours prior to admission, when the temperature rose to 102 F. and irritability and difficulty in breathing developed.

On admission, this infant appeared toxic and had marked dyspnea with inspiratory stridor and reversed thoracic breathing. The pharynx and the tonsils were injected. The remainder of the physical examination was essentially negative.

The infant was placed in a croup tent and was given 1 grain (0.06 Gm.) of sulfanilamide per pound of body weight. Nose and throat cultures were negative for diphtheria bacillus. The temperature returned to normal on the seventh day in the hospital, and the child appeared much better, but the respiratory stridor continued for several days.

Direct laryngoscopic examination was performed fifteen days after admission, and the following note was dictated: "Epiglottis has a characteristic omega shape of small children, but the aryepiglottic folds are particularly large and still somewhat thickened, although the acute inflammatory process has completely subsided. The thickening and increased size of these folds is carried backward to the arytenoids which are also slightly larger than normal. The vocal cords are normal."

It was the impression of the laryngoscopist that the child had laryngomalacia (or hypodynamia laryngis) which resulted in stridor in the presence of infection of the upper part of the respiratory tract.

The infant was seen in the outpatient department a month later and was symptom free.

Case 3.—P. B., a 4 month old white boy, was admitted to the hospital on Feb. 17, 1940 and was discharged on February 24. Ever since birth, the mother had noticed that the infant had a high-pitched cry and that it seemed short of breath at irregular intervals. The mother was told by the family physician that the infant had an enlarged thymus which was treated by roentgen therapy without improvement.

A few days before admission, mild infection of the upper part of the respiratory tract and marked inspiratory stridor developed, but no cyanosis.

On admission, the temperature was 101 F., and the infant was moderately toxic. Physical examination disclosed an injected pharynx and the inspiratory stridor

but no other significant findings. The temperature became normal on the third day, but stridor was still present.

Laryngoscopy was performed a week after admission: "The aryepiglottic folds hung loosely over lateral laryngeal walls. During inspection, the actual collapse of the laryngeal vestibule could be observed, showing the epiglottis sucked into the glottic chink. It was with difficulty that the epiglottis could be picked up on the tip of the laryngoscope, but when this was done, the larynx, except for a moderate degree of redness, was found to be normal."

These cases were reported by courtesy of Dr. Reuel A. Benson, Fifth Avenue Hospital, and Dr. Herbert Berger, Richmond Memorial Hospital.

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ELECTROAUDIOGRAPHY

ANALYSIS AND INTERPRETATION OF THE AUDIOGRAM

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The increasing use of the electroaudiographic examination by nonmedical agencies, as well as by the medical profession, warrants a discussion of the actual value of the electroaudiogram in the diagnosis of hearing deficiencies. Knowledge of otologic as well as cardiac problems has been added to greatly by the results of the electrical methods in recording otologic and cardiac pathology. The diagnostic value of the electroaudiogram in many ways parallels that of the electrocardiogram.

Electroaudiography, undoubtedly the most revealing of the many mechanical aids in otology, provides a means of determining numerous changes in the neural elements and conduction system of the acoustic apparatus which other physical diagnostic measures cannot accurately detect.

The electric audiometer was originally devised about 1920 as the result of research work endeavoring to formulate a theory of telephone speech quality. In order to determine the degree of the excellence of speech when transmitted over an electrical system, it was necessary to evaluate the human element concerned. This valuation consisted in measuring and carefully analyzing speech and hearing, which is the receptor. To accomplish this the electric audiometer, a by-product of the telephone industry, was devised. However, the appearance of the electric audiometer in 1925 as a diagnostic aid in acoustic problems produced little enthusiasm among otolaryngologists, as it was soon discovered the instrument failed to live up to its early predictions in many respects. Further research, especially that concerning the development of the vacuum tube oscillator, finally produced the modern electric audiometer, which consists of three essential elements: the oscillator, the attenuator and the receiver.

In the interests of clinical efficiency, arising from the increasing use of the audiometer by nonmedical groups and by the medical profession, a standard of requirements for the instrument was formulated. This was necessary, as the degree of accuracy of the numerous instruments

From the Department of Otolaryngology of the Alfred Benjamin Dispensary.

on the market varied greatly. The Committee on Standards of the American Medical Association set up a code of requirements for electric audiometers. Only a few of the instruments on the market at present meet these requirements. Audiometers failing to meet these standards can hardly be relied on to maintain a reasonable degree of accuracy and stability. These considerations are of prime importance, as statistical data pertaining to hearing should be measured by the same scientific yardstick, a standardized instrument and technic, so that all electroaudiograms will be comparable.

The use and abuse of the electroaudiogram by various nonmedical agencies, which are entirely unfamiliar with otologic pathology, have brought the need for a better understanding of electroaudiography among those who possess the necessary background to investigate the otologic problems of deafness and of hearing deficiencies.

The basis of the electroaudiographic examination is the stimulation of the perceptive mechanism by pure tones of desired frequencies, namely, those sounds necessary in one's daily occupation. This is accomplished by utilizing electrical sound in the form of an electric current from which all extraneous components have been removed. The electrical sound, or pure tone, is passed into a receiver, where it is converted into acoustic energy to stimulate the hearing apparatus. By this method it is possible not only to measure the threshold of hearing but to analyze the entire area of audibility with much precision.

In measuring the acuity or deficiency of acoustic perception, one is concerned with two factors in testing: intensity, or loudness, and frequency of vibration, or pitch, as sensed by the hearing mechanism. The normal auditory range of the human ear extends from the 16 to the 32000 cycle note. However, few adults, especially those in later life, are able to perceive a 16000 double vibration note. For all practical purposes this is not necessary, as few of the sounds in routine daily life exceed a pitch of 10000 cycles. Yet in order to obtain a complete picture of a subject's acoustic status it is necessary to test the entire 120 to 12000 cycle range. If this procedure is faithfully followed, hearing defects beginning in the frequencies above the speech range, which otherwise might not be diagnosed or suspected, will be detected. This is seen so frequently in the electroaudiogram as to justify regular periodic aural health examinations.

The value of the electroaudiogram in diagnosis is not fully appreciated until the ability to interpret accurately the findings is acquired. The analysis of a person's acuity or deficiency of hearing is based on certain quantitative and qualitative measurements of sound perception. These data can be obtained accurately only through electroaudiography.

The sensitivity of the perceptive mechanism varies with the different frequencies. The ear is much more sensitive in the middle of the pitch range, where the centers of speech exist. In other words, the ear is not equal in its response or sensitivity throughout the various frequencies. However, in an electroaudiographic examination one is not concerned with the relative sensitivity of the perceptive mechanism at the various frequency levels. The audiographic measurements deal with the variations from normal sensitivity at each level and not with their relative sensitivity. Therefore, on the audiogram a straight isoelectric line represents the normal threshold of audibility. Any variation below the isoelectric line indicates a defect in hearing. Many types of hearing deficiencies assume characteristic variations from the isoelectric line. These variations produce audiographic patterns and form the basis on which the various types of acoustic impairment are detected and differentiated. Nevertheless, the audiogram does not replace the important procedures of history taking and physical diagnosis, since some acoustic abnormalities which are the result of more than one etiologic factor may not produce typical audiographic changes. In addition, variations occur in the audiographic pattern depending on the duration of the lesion.

The normal electroaudiogram consists of a series of sensation points as represented by the isoelectric line. Any deflection above the normal threshold represents a superacuity in hearing. Deflections below the line indicate acoustic defects. The patterns which they form enable one in the majority of cases to make an accurate differential diagnosis and an estimation of acoustic damage. In addition, facts concerning prognosis and measurements of functional response are obtained. Consideration of these factors is necessary in determining the therapeutic approach to the clinical problem of hearing deficiencies. The electroaudiographic method has rationalized this therapeutic approach.

In summary, the chief uses of the electroaudiogram are: (1) the detection and differentiation of acoustic deficiencies; (2) the recognition of obscure perceptive defects; (3) the measurement of functional response; (4) the estimation of therapeutic effects; (5) the observation and recording of the progress of the hearing condition; (6) the provision of definite data in documentary form, as required in medicolegal cases dealing with hearing deficiencies resulting from industrial causes, and (7) the prescription of hearing aids.

A normal electroaudiogram is of definite value in ruling out a hearing deficiency in the presence of questionable symptoms. Often mental and behavior problems of the school child are obscured by an apparent loss of hearing which in reality does not exist. Likewise, an abnormal

audiogram often reveals that a behavior problem is due to a true deficiency in hearing.

The detection of acoustic deficiencies should occur long before serviceable hearing is impaired. Methods other than electroaudiography are unreliable and fail to reveal early defects in the hearing mechanism. If serviceable hearing is to be preserved the detection of acoustic deficiencies must occur early in the course of the disease. In the majority of cases the progress of hearing defects can be arrested in their early stages. Coates [1] strongly emphasized that the detection of incipient losses of hearing is of utmost importance if restoration to normal is to be obtained or if progression to an incapacitating degree of deafness is to be avoided. From this standpoint alone, electroaudiography supersedes all other diagnostic methods in accuracy. The older methods in testing for hearing deficiencies have too many variable factors to insure reliable results. In the majority of cases incipient hearing defects can be detected only by electroaudiography. This fact justifies its use in the diagnosis of every case in which a hearing loss is suspected.

The differentiation of acoustic deficiencies is made possible by the characteristic tracings which are formed on the audiogram. From the audiographic pattern one can readily discern the type of hearing defect. This applies not only to the general classification of the hearing defect but more precisely to the possible etiologic background.

The audiographic findings discussed here are based on the perception of sound by air conduction, the normal mode of hearing. In the audiographic tracings, frequency losses not exceeding 30 decibels up to 1024 cycles indicate damage to the conductive mechanism, whereas losses above 1024 cycles and those exceeding 30 decibels below 1024 cycles indicate damage to the perceptive mechanism. The audiographic patterns of the various types of conductive deficiencies are similar. The acoustic defects occur in the 128 to 1024 cycle range. The frequency losses in purely conductive defects rarely exceed a loss of 20 to 30 decibels in this range. The patterns are usually regular and do not show abrupt or marked deflections from the isoelectric line (fig. 1).

Audiograms which show an average loss exceeding 25 to 30 decibels in the conductive range invariably show losses in the perceptive range, 1024 to 11584 cycles. Conditions such as this with defects in both the conductive and the perceptive range are classified as a defect of mixed type, or deafness depending on the degree of hearing deficiency (fig. 2).

A type of conductive deafness which produces an entirely different audiographic pattern is frequently seen in children who have an excessive

1. Coates, G. M.: The Prevention of Deafness, Ann. Otol., Rhin. & Laryng. **40**:651 (Sept.) 1931.

amount of lymphoid tissue obstructing the orifices of the eustachian tubes. The audiogram shows losses in the perceptive range with relatively no losses in the conductive range (fig. 3). This condition was originally described by Crowe and Baylor.[2] The loss in the perceptive range in this condition does not necessarily imply an inner ear or nerve lesion, which one would naturally assume from the audiographic pattern. Sturm,[3] in a study of several hundred patients with deafness, showed

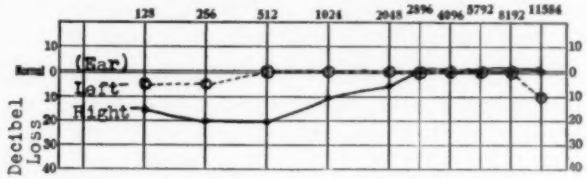

Fig. 1.—Auditory defects in conduction deafness.

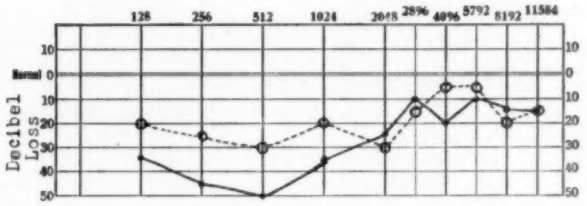

Fig. 2.—Acoustic defects in the mixed type of deafness.

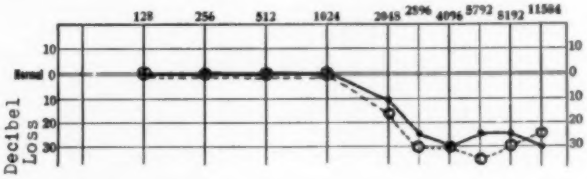

Fig. 3.—Auditory defects in adenoid deafness in children.

that practically one third owed their hearing disability to this type of nasopharyngeal disease in childhood.

Acoustic defects of the perceptive type occur more frequently than those of the conductive type. Focal and systemic sources of infection are responsible for the majority of perceptive defects. Focal infections

2. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. **112**:585 (Feb. 18) 1939.

3. Sturm, A. P.: Adenoid Deafness, Ann. Otol., Rhin. & Laryng. **37**:579 (June) 1928.

of the gastrointestinal, the genitourinary and the lymphatic system, as well as the upper respiratory tract, produce the major portion of perceptive defects in the acoustic mechanism. Often the presence of perceptive defects is unknown to the patient. The hearing loss occurs in the upper limits of and above the serviceable hearing range (fig. 4).

The recognition of obscure perceptive defects is of utmost importance if the integrity of the auditory mechanism is to be preserved. Since most perceptive defects are progressive, their early detection will do much to prevent irreparable damage to hearing. The positive diagnosis of an early or incipient auditory defect usually cannot be made without the aid of the electroaudiogram, and the audiographic tracings give the best means not only of observing progress but of permanently recording it.

Focal and systemic infection in the early stages of acoustic impairment produces defects in the upper limits of the perceptive range. This may be due to a difference in sensitivity of the physiologic mechanisms

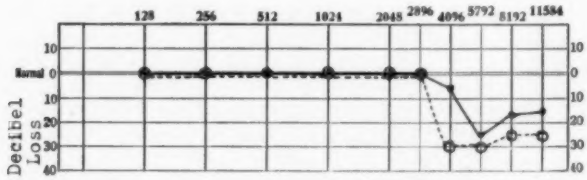

Fig. 4.—Early perceptive defects not affecting serviceable hearing.

controlling the different frequencies. Apparently the mechanism controlling the higher frequencies is more delicate than that controlling the lower frequencies; and hence is more sensitive to toxic invasion. Perceptive deafness or defect in hearing does not necessarily imply a nerve lesion, as is commonly believed. Perceptive defects may arise from a number of disturbances in the cochlear mechanism. Oda [4] demonstrated cochlear lesions in which various degrees of atrophy of the nerve, the organ of Corti, the external sulcus cells and the stria vascularis had produced acoustic defects. Babbitt [5] stated that syphilis of the central nervous system produces cochlear degeneration by cutting off the blood supply rather than by affecting the auditory nerve. Mosher [6] showed

4. Oda, D.: Observations on the Pathology of Impaired Hearing for Low Tones, Laryngoscope **48**:786 (Nov.) 1938.

5. Babbitt, J. A.: A Study of Medical and Surgical Aids to Hearing, Surg., Gynec. & Obst. **68**:395 (Feb. 15) 1939.

6. Mosher, H. P.: Does Animal Experimentation Show Similar Changes in the Ear of the Mother and Foetus After the Ingestion of Quinine by the Mother? Laryngoscope **48**:361 (June) 1938.

that the action of certain drugs, i. e., sodium salicylate, quinine and mapharsen, may produce congestion and hemorrhage of the vessels of the cochlea. Selfridge [7] stated that the hearing defects of old age are the result of trophic disturbances affecting the cochlea. Mellanby [8] demonstrated that a diet deficient in vitamin A produced degenerative changes in the cochlear nerve, the cells of the spiral ganglion and their central and peripheral branches. McCord and associates [9] stated that hearing defects arising from industrial noises are due to damage of the organ of Corti. These are only a few of the pathologic conditions of the cochlea which produce perceptive defects in hearing. In brief, disturbances in the cochlear mechanism may arise from various neural defects; from vascular lesions, i. e., arteriosclerosis, syphilis, hyperemia, hypertension and hemorrhage; from acute infectious diseases involving the membranous cochlea, and from diseases which interfere with the normal function of the intralabyrinthine fluid, the final common pathway of sound conduction. The differences in cochlear lesions depend on the structure or structures involved, the degree of involvement and the amount of secondary atrophy. An understanding of the pathologic changes occurring in the cochlea and their etiologic background is necessary before a rational therapeutic approach to the problem of arresting and correcting a hearing deficiency can be made.

The audiographic patterns of the various types of perceptive deficiencies are similar only in that the defects are mostly confined to the frequencies above 1024 cycles. The numerous variations which occur in cases of these high frequency defects are characteristic of different types of auditory deficiency. Information concerning the cause of the hearing impairment, the activity of the pathologic process and the duration of the acoustic lesion can be derived from the character of the audiographic tracings. Perceptive defects originating from a focus of infection produce an audiographic pattern in which the losses are usually abrupt and progressively irregular. Regressions occur but rarely reach the isoelectric line (fig. 5).

Often stair step defects are observed in which successive frequencies show the same amount of loss. The irregular defects of early perceptive deafness gradually disappear the longer the focal source of infection exists. The audiographic pattern in the later stage of acoustic impairment shows a gradual and progressive depression of the higher

7. Selfridge, G.: Chronic Progressive Deafness from a Nutritional Stand-Point: A Preliminary Report, Ann. Otol., Rhin. & Laryng. **46**:875 (Dec.) 1937.

8. Mellanby, E.: The Experimental Production of Deafness in Young Animals by Diet, Laryngoscope **49**:1090 (Nov.) 1939.

9. McCord, C. P.; Teal, E. E., and Witheridge, W. N.: Noise and Its Effect on Human Beings, J. A. M. A. **110**:1553 (May 7) 1938.

frequencies. Focal infection of long duration may show conductive defects in addition to the perceptive defects in the audiogram (fig. 6). The involvement of the conductive range in focal infection indicates an advanced degree of disturbance in the hearing mechanism. With these changes the prognosis should be guarded.

As stated by Coates and Gordon,[10] when a patient himself is aware that a hearing loss exists, often irreparable damage has occurred. Too much emphasis cannot be placed on the early detection of hearing impairments before practical hearing is involved. The routine analysis of the frequencies above the speech range is, therefore, very important. While the loss of pure tone perception at these frequencies does not necessarily impair serviceable hearing, it forewarns of a progressive

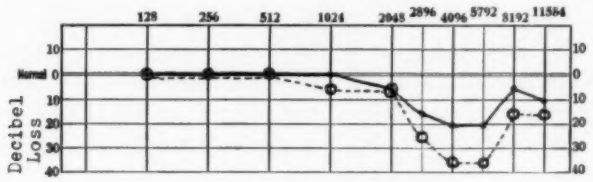

Fig. 5.—Perceptive hearing defects associated with focal infection.

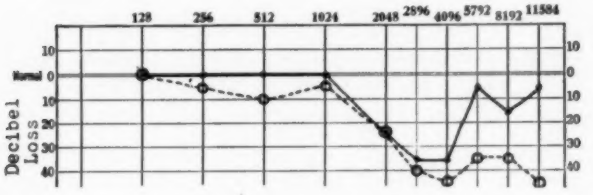

Fig. 6.—Acoustic defects in the late stage of focal infection.

process which will eventually involve the speech range. When dip and peak formations are observed in the high frequency range of the audiogram, early toxic invasion of the acoustic mechanism should be suspected. With these findings therapeutic measures can be instituted early enough to arrest and control the infection, so that the viability of the hearing apparatus is preserved.

Syphilitic infection produces perceptive defects which are similar to those of focal infection. The perceptive defects of syphilitic infection, however, differ from those of focal infection in that the abrupt losses are more irregular. Regressions are common and often reach the iso-

10. Coates, G. M., and Gordon, W.: The Otologic Management of Progressive Deafness, Tr. Am. Acad. Ophth. **38**:309, 1933.

electric line (fig. 7). Very early syphilitic defects may show only a single frequency deficiency, with no other loss in the entire range. As pointed out by Babbitt,[5] the deficiency is probably due to a disturbance in the blood supply to the cochlea rather than to an auditory nerve defect. As in focal infection, the audiographic pattern observed in the later stage of syphilitic infection shows a gradual and progressive loss in the higher frequencies instead of the irregular pattern seen in the early stage of acoustic impairment.

Occasionally unknown congenital hearing defects are manifested by a single high frequency defect (fig. 8). These are differentiated from syphilitic defects by serologic tests and repeated audiographic studies. Congenital defects of this nature are nonprogressive and rarely impair

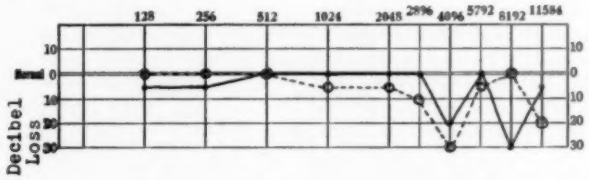

Fig. 7.—Perceptive hearing defects associated with syphilitic infection.

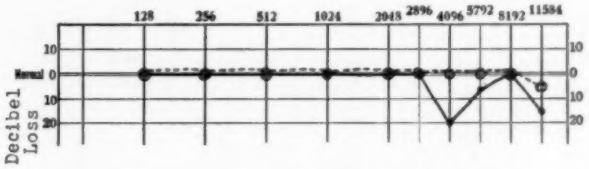

Fig. 8.—Audiographic pattern associated with a congenital hearing defect.

hearing, since they involve the frequencies above the serviceable hearing range.

The auditory losses of presbycusis, when first noticed by the patient, usually occur at and above the 2048 cycle note. Up to 70 years of age hearing for the frequencies below 1024 cycles remains practically normal. At 50 to 60 years of age there is usually some involvement of the frequencies above the 2048 cycle frequency (fig. 9). The higher frequencies show the greater loss, with progressive involvement of the middle and the lower end of the scale if the impairment continues. Abrupt and irregular losses in the audiographic pattern are rarely observed. The perceptive defects of presbycusis form a gradual and progressive loss for high to low frequencies in the audiogram. The auditory loss in presbycusis is apparently the result of a trophic disturbance in the neural elements of the cochlea.

Otosclerosis presents several variations in the audiographic pattern. The character of the acoustic defects depends on the length of time the hearing loss has been apparent to the patient. In the earlier stages the hearing loss results from impairment in the action of the stapes in the oval window articulation. The restricted action of the stapes interferes with the normal function of the intralabyrinthine fluid, the final common pathway of sound conduction. In the later stages secondary changes in the vascular and neural elements of the cochlea produce marked perceptive defects.

The audiographic pattern in the earlier stage of otosclerosis shows practically the same amount of loss in the higher as in the lower fre-

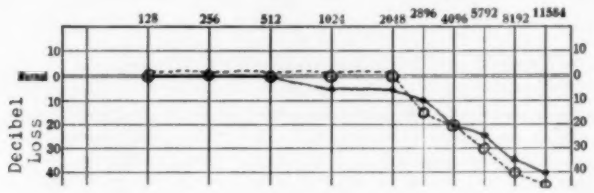

Fig. 9.—Auditory defects of presbycusis.

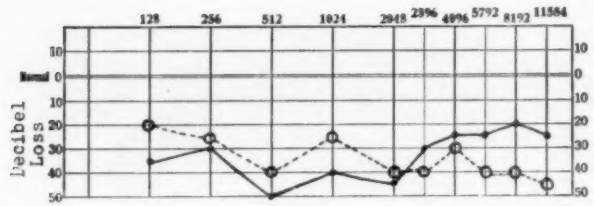

Fig. 10.—Auditory defects in early otosclerosis.

quencies. The dampening effect of the ankylosing foot plate of the stapes prevents normal intralabyrinthine fluid displacement, with the result that the transmission of sound is interfered with throughout the entire cochlea. This mechanical interference suppresses the perception of all tones, high and low (fig. 10).

The auditory defects of early otosclerosis occur throughout the entire auditory range. The losses are not irregular. The perception for pure tones is reduced more or less equally for high as well as low tones. Crowe [11] demonstrated these audiographic features in cases of early uncomplicated otosclerosis. As the spongiosis of the labyrinth progresses the vascular and neural elements of the spiral lamina are involved. This

11. Crowe, S. J.: Diagnosis and Differential Diagnosis of Deafness, Arch. Otolaryng. **28**:663 (Nov.) 1938.

later stage of otosclerosis is manifested by an increasing loss for high tones in the audiogram. The greater loss of high tones in the late stage is probably due to the extension of the spongiotic process from the site of predilection, the stapediovestibular joint, to the adjacent basal coil of the cochlea, where the high tone mechanism is located (fig. 11).

Electroaudiography provides the means by which data in documentary form can be furnished in medicolegal cases dealing with hearing defects resulting from trauma. From the medicolegal standpoint the investigation of an auditory loss resulting from trauma requires definite data as to the amount of hearing loss in each ear, the differentiation of complicating acoustic deficiencies and the prognosis. Audiographic study provides the major portion of the information desired in these cases.

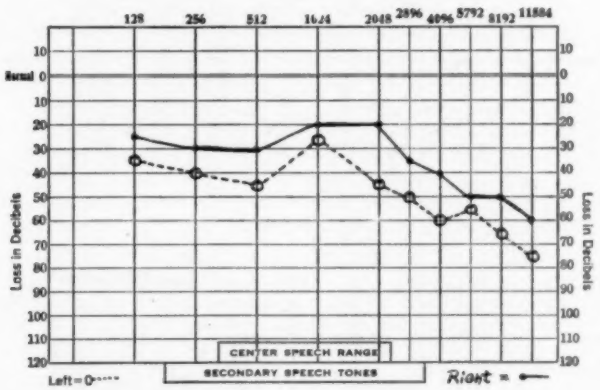

Fig. 11.—Auditory defects in late otosclerosis.

Traumatic deafness may be roughly divided into three types: occupational, detonation and aerovibration deafness. Occupational deafness results from prolonged exposure to industrial noises. The acoustic defects are due to a noise environment in which there is more or less a constant din. The condition is chiefly observed in boiler makers, steel workers and construction workers. The constant bombardment of the acoustic apparatus by the mixture of high and low tone noises occurring in these occupations produces a degeneration of hearing acuity, first in the middle and then in the upper limit of the auditory range (fig. 12). The audiographic defects are usually bilateral and follow the same general outline. However, the patterns rarely coincide, as the defects are more pronounced in one ear.

It must be remembered that traumatic deafness may arise from the prolonged exposure to any type of industrial noise. The nature of the working conditions under which many of these noises occur hinders

the application of preventive measures. In order to avert medicolegal entanglements frequent audiometric examinations should be required of persons in these occupations, so as to detect those who are especially susceptible to traumatic deafness. However, not all persons are affected to the same extent in a common noise environment. Many have been known to work for years in a noise environment with relatively little impairment in hearing. Persons who show an early tendency to acquire hearing defects from industrial noises should be transferred to less harmful environs.

Detonation deafness is caused by a sudden loud noise near the ear. There may or may not occur a rupture of the tympanic membrane with this trauma. Owing to the character of the sound the hearing defect

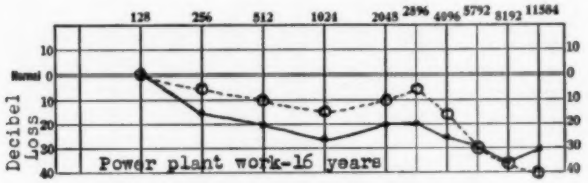

Fig. 12.—Audiographic defects of occupational deafness.

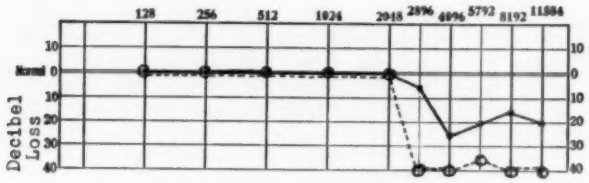

Fig. 13.—Auditory defects of detonation deafness.

is usually more marked in one ear. A bilateral hearing deficiency, in which the defects are similar and show approximately the same amount of loss, rarely results from this type of injury. Vestibular examination should always supplement audiographic studies in cases of this type. Detonation deafness is often accompanied by a disturbance in the kinetic-static mechanism. The acoustic defects produced by a sudden loud noise may occur in all frequencies but are observed usually in the high frequencies at and above 4096 cycles.

A sudden loud noise causes more impairment in hearing for high than for low tones. Guild [12] stated that impairment in hearing is usually

12. Guild, S. R.: War Deafness and Its Prevention, Ann. Otol., Rhin. & Laryng. **50**:70 (March) 1941.

limited to frequencies above 2048 cycles if recovery occurs. However, a single detonation can cause permanent severe impairment of hearing for all tones. Tinnitus practically always accompanies this type of traumatic deafness. The majority of traumatic hearing defects tend to improve with time. Periodic audiographic studies for approximately one year are necessary to determine the final integrity of the auditory mechanism.

A third type of traumatic deafness, aerovibration deafness, has come to the attention of otologists during the last few years because of the increasing amount of military, commercial and private air traffic. Campbell and Hargreaves [13] demonstrated the initial effect to be evident at the 4096 cycle frequency (fig. 14). Continued exposure to the combination of vibration and noise deepens and spreads fanwise the 4096 notch, so that eventually the speech range is encroached on, with the production of conversational deafness. Apparently, some persons are not affected by long exposure to this environment and after years of

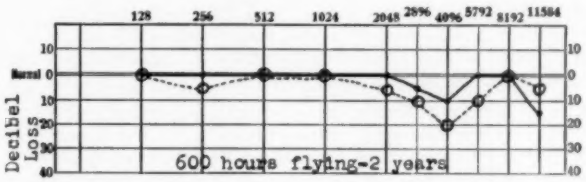

Fig. 14.—Audiographic defects in aerovibration deafness.

flying show little impairment in hearing. Hearing defects due to aerovibration should not be confused with those due to aero-otitis media. The audiographic defects in aero-otitis media involve the low frequency range, as in conductive deafness. The hearing impairment in this condition is due to changes in atmospheric pressure which interfere with the ventilation of the middle ear, producing discomfort, pain, congestion and occasionally rupture of the tympanic membrane.

Impairment in hearing is not uncommonly associated with injury to the head. The degree and type of loss depend on the structures involved and the severity of injury. The audiographic pattern may show a high or low frequency deficiency or a combination of the two. Repeated audiographic studies are necessary to determine the final integrity of the auditory mechanism in this condition.

The measurement of functional response is obtained by testing pure tone perception throughout the entire range of hearing. An accurate and detailed study of the functional status of the constituent parts of

13. Campbell, P. A., and Hargreaves, J.: Aviation Deafness, Arch. Otolaryng. **32**:417 (Sept.) 1940.

the hearing apparatus can be made only by electroaudiographic examination. By testing the individual frequencies it is possible not only to measure the threshold of hearing but to analyze the entire area of audibility with much precision. By this method word deafness can be distinguished from physiologic deafness. Word deafness occurs in certain neurologic conditions in which the higher brain centers are involved. In cases of this type the loss for pure tone is usually very slight, and hence physiologic deafness can be ruled out. This method is especially useful in distinguishing a true physiologic loss from lack of development of a sound vocabulary.

The prognosis of the hearing condition is determined to a great extent by the measurements of the functional response. A guarded prognosis should be given when there is a total loss in any one of the

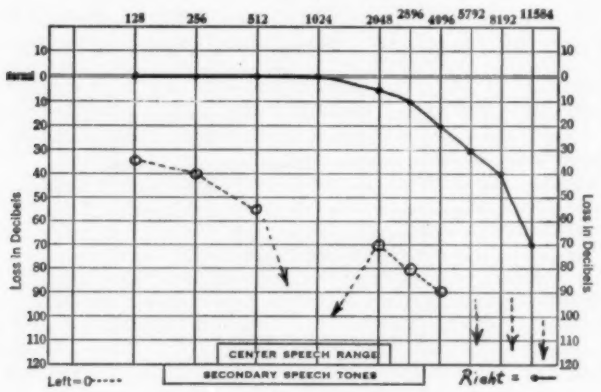

Fig. 15.—Audiographic defects indicating a poor prognosis.

frequencies or when the average loss in the speech range exceeds 30 decibels (fig. 15).

The amount of pure tone loss, both individual and average, in the speech range, the character of the audiographic pattern of the pure tone losses throughout the entire area of audibility and the distortion in perception of the pure tones give many helpful hints as to the prognosis of the hearing condition.

Although the expression of functional response in terms of percentage loss is sometimes considered undesirable, this form of presenting data meets the requirements necessary in medicolegal cases. In addition, the recording of the hearing defect in documentary form furnishes definite evidence concerning other pertinent factors which are indispensable in the final evaluation of the auditory condition. The advantages of the electroaudiographic method of acoustic analysis are

quite evident in determining the therapy in hearing problems. The effectiveness of any form of therapy can be definitely ascertained by the comparison of audiograms made at intervals during the time of treatment. By this method the progress of the hearing condition can be determined at any time, and detailed recordings for future reference can be secured. In prognosis the tracings are of value in revealing new lesions and in showing progressive changes.

The mechanics of an audiometric examination should not be regarded too lightly. The numerous physical and psychologic factors involved greatly influence the recording of the audiogram. Their proper evaluation is of utmost importance during the examination. The rate of fatigue for individual frequencies during testing, the distortion of pure tone perception, particularly in the higher frequencies, the application of masking technic, the determination of the noise threshold and many other pertinent factors affect the audiographic recording and influence the interpretation of the audiogram. It is understandable, then, that a certain amount of skill and knowledge is necessary in the operation of the electric audiometer. To relegate the examination to assistants, particularly to nonmedical agencies, invites inaccuracy in testing and in the subsequent interpretation of the findings. The present lack of personal interest evidenced by the otologist in electroaudiography is giving rise to a group of so-called audiometrists who are in no way prepared intelligently to investigate and advise a patient concerning his hearing problem. As pointed out by Newhart and Hartig,[14] the development of the audiometer has made it possible for hearing aid distributors, under the title of "consultant in hearing aids" or "audiometrist," to disturb the close relation between physician and patient. Because of their limited understanding of otologic problems, these persons in many cases have unknowingly misled the patient into the belief that a hearing aid would solve his hearing problem. Audiometric analysis, as well as any other mechanical method of diagnosis, is incomplete without a thorough physical examination. In addition to the audiographic examination, the clinical investigation of the ear, nose and throat is absolutely necessary before an opinion on the plan of therapy can be formulated. This justifies the impression that the prescription of hearing aids should be issued only on the advice of the otologist.

Electroaudiometric speech tests with the air and bone conduction apparatus, supplemented by audiographic analysis, offer the most accurate means at present by which the otologist can give advice con-

14. Newhart, H., and Hartig, H. E.: Hearing Aids, Arch. Otolaryng. **29**:614 (April) 1939.

cerning hearing aids. The air and bone conduction audiograms, together with the results of the audiometric speech perception tests, enable the otologist to determine in the majority of cases whether a bone or an air conduction type of hearing aid should be used. One of the more important services the otologist renders in the selection of a hearing aid is to enable the patient to select an instrument without the interference of one who is interested in its sale.

In summary, electroaudiography justifies its place in diagnosis because of its accuracy and because of its value in differential diagnosis, in the determination of acoustic damage, in the estimation of therapeutic results, in the measurement of functional response, in the detection of incipient hearing defects and in prognosis. This graphic method has standardized diagnosis, simplified prognosis and rationalized therapy in the problem of defective hearing.

608 Commerce Building.

TONSILLECTOMY AND ITS EFFECT ON THE SINGING VOICE

WILLIAM A. C. ZERFFI
NEW YORK

The question as to whether the singing voice is beneficially or adversely affected by removal of the faucial tonsils is of grave importance to singers. A singer whose tonsils are in such an infected condition as to be a menace to his general health, yet whose livelihood depends on his voice is faced with a decision which may well give him cause for worry. While he may know singers who have been benefited by this operation, he also probably knows or has heard of those who have suffered definite impairment of their vocal resources as the result of tonsillectomy.

As is usually the case in all matters pertaining to the singing voice, accurate data are extremely difficult to obtain, and this is particularly true in regard to the question which I discuss. There is no doubt that in earlier days much damage was done to singers' throats by the removal of tonsils, especially when complete removal, as opposed to partial removal, first began to be practiced. However, with the greatly improved technic in use, this danger has been reduced so greatly that it is unusual to find a throat which has suffered serious damage.

Unfortunately, the function of the soft palate in voice production is rarely understood by vocal teachers, and the same can be said, though in lesser degree, of laryngologists. Many laryngologists are frank to admit that voice production is out of their sphere, and I have often regretted that this is so. Valuable voices might have been saved had their owners been able to rely on the advice of laryngologists who were familiar with the physiology of voice production instead of having to rely on vocal teachers and the voodoo rites of conventional voice teaching.

According to Hudson Makuen, the soft palate has two important functions in voice production: (1) its valvular function in influencing vocal resonance and (2) its power of tilting the thyroid cartilages, thereby stretching the vocal cords and in this way influencing the quality and the pitch of the voice. It would be difficult to find a more accurate or simpler description of the action of the soft palate or one which more clearly emphasizes the dangers which lie in any damage to the soft palate by an improperly performed tonsillectomy. Two muscles of the utmost importance to the voice lie behind the tonsils, the palatopharyngeus and the stylopharyngeus. These two muscles are attached to the

rear of the thyroid cartilage, and it is their action which tilts the cartilage and helps to tense the vocal cords. Any injury to these muscles is disastrous to the production of the voice, since it destroys the higher tones. This view regarding the importance of the action of the soft palate has more recently been confirmed by Dr. Lee M. Hurd, of New York. It has been my good fortune to be able to consult with Dr. Hurd over a period of some twenty years and to receive his invaluable counsel and advice. He has many times displayed an almost uncanny accuracy in the diagnosis of singers' troubles, muscular as well as pathologic, due to his keen interest and comprehensive knowledge of vocal physiology. In regard to the after-treatment following tonsillectomy, Dr. Hurd insists that there be complete cessation from singing for at least four to six weeks. As he explains it, the entire palatal area is likely to be involved when infected tonsils have been present. Adequate time must therefore be allowed for the infection to subside and the muscles to return to a more or less normal state before any attempt is made to sing. A singer is also advised to begin with very light exercises and to rest frequently, so that muscle tone may be restored gradually to the palatal muscles and the formation of scar tissue reduced. Attempts to make full use of the voice before the throat is thoroughly healed may lead to irreparable damage. In this connection one of the most interesting cases which has ever been brought to my attention was that of a young woman whose soft palate had been virtually destroyed, nothing having been left but a tight band of scar tissue, which was practically immobile. She possessed a voice of pleasing quality in the lower and medium ranges, but high tones could be produced only by forcing the vocal cords together with the help of the external muscles. Any prolonged attempt to sing high tones invariably resulted in the formation of vocal nodules. So long as the voice was used in the lower registers, no difficulty was experienced.

So much for the results of poorly performed operations, which, as has been suggested, are relatively rarely encountered.

However, despite improved technics for the performance of tonsillectomies, one still hears of instances in which a singer's voice seems to have been adversely affected by the operation, and yet examination does not reveal the slightest lesion of the palatal muscles. It is such instances which give a singer cause for agonizing worry and apprehension. He knows of cases in which the surgeon was as skilful as is humanly possible, and yet trouble resulted for the patient. Surgeons are only too well aware of this, and sometimes, when the singer has enjoyed a wide reputation, they hesitate and have been known to refuse to operate for fear that vocal impairment might result.

This seeming paradox, however, can be explained by recognition of the fact that some temporary impairment of the palatal muscles after tonsillectomy is unavoidable. But it cannot be too strongly emphasized

that this impairment need be only temporary and can be eliminated in a relatively short time provided correct vocal exercises are employed. No matter how carefully and efficiently the operation is performed, a certain amount of scar tissue forms and some stiffness in the action of the palatal muscles must be expected. Every singer, no matter how badly trained he may have been, has a certain amount of development of the palatal muscles. Singers who have had the advantage of sound training will be found to have greater strength and flexibility in these muscles and are therefore in a better position to overcome any temporary handicap which may occur after tonsillectomy. But a singer who forces his voice and employs the type of voice production which was outlined by me in an article published elsewhere [1] will find that the loss of this palatal action will cause him to depend more and more on the external (swallowing) muscles for the production of tone and will inevitably find himself in serious vocal difficulties. When this occurs, it will be believed that the operation was the direct cause of the trouble, even though the surgical treatment was successful. Consultation with other surgeons will yield no more than a confirmation of the latter fact. The singer finds himself in a cul-de-sac, from which escape seems impossible and often is. I have observed a number of cases in which the voices of persons who were singing successfully despite poor voice production declined with amazing rapidity after tonsillectomy, despite the fact that the operation was skilfully performed.

Such tragedies could be avoided if singers were taught how to stimulate the action of the palatal muscles. These are the only vocal muscles which are in any way capable of being consciously affected. All others operate subconsciously, but a singer can be trained to raise or lower the palate at will. Since the normal swallowing action brings about a raising of the palate and a drawing down of the tongue, this dual action must literally be broken up in order to allow the palate to operate independently of the tongue. If a singer does not learn to disassociate the action of the palate from that of the tongue, the lowering of the tongue will interfere with the free action of the larynx. And it need hardly be said that freedom of action of the larynx is a prime requisite for correct voice production.

The conclusion is unavoidable that accurate knowledge of the action of the palatal muscles is of inestimable advantage both to singers and to laryngologists. As the matter stands, in the majority of cases singers are faced with a decision which may well give them cause for apprehension. Their health demands tonsillectomy, yet they have a real and unfortunately justified fear that their voices may be adversely affected. It is a situation which should not be allowed to persist.

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EXPERIMENTAL HYPERSENSITIVITY OF MUCOUS MEMBRANES OF THE UPPER RESPIRATORY TRACT

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The problem of the "allergic mucous membrane" constantly confronts the rhinologist. Many of the properties of the mucous membrane are poorly understood, such as its susceptibility to infection and how important a factor bacterial hypersensitivity is. Preliminary to an experimental investigation of some of these relatively complicated problems, means and methods of sensitizing the mucous membrane were studied. The purpose of this report is to present the results of our observations on experimental sensitization of the mucous membrane of the upper respiratory tract.

Several investigators have reported successful sensitization of mucous membranes by local applications or by injections of an antigen or antibody.[1] Our experiments disclose that the mucous membrane of the upper respiratory tract can be readily sensitized by various indirect routes. Moreover, animals can be systemically sensitized through the mucous membrane of the sinus, the mucous membrane being left intact.

METHODS

Half-grown white rabbits weighing from 1 to 1.5 Kg. were used in these experiments. The antigen was a 5 per cent aqueous solution of egg white. This substance was chosen because it is known to produce reliable sensitization. Bacterial antigens were found to be less dependable.

This study was aided by a grant from the Joseph G. Snydacker Fund.

From the departments of otolaryngology, pathology and bacteriology of Michael Reese Hospital.

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The marginal vein of the ear was the site of the intravenous injections. The subcutaneous, intracutaneous and intraperitoneal injections were performed into or through the abdominal wall, after the hair had been thoroughly removed. One cubic centimeter of aqueous solution of egg white was used for each injection, and the injections were made at weekly intervals for from three to five weeks. The best sensitization was obtained with five injections.

Before any injections into the sinuses of living animals were made, the sinuses of several dead rabbits were dissected. The rabbit's maxillary sinus is apparently analogous to the antrum of the human being. The lateral wall of this sinus is perforated, the fenestra being sealed by a soft tissue membrane. With only slight pressure, a 20 gage needle can be introduced through this fenestra into the sinal cavity. It was further observed that the wide connection between the nasal cavity and the maxillary sinus permits any fluid injected into the sinus to flow immediately into the naris. In an attempt to retain the antigen within the sinus for a longer period, the antigenic fluid was thickened with agar. This material, stained with blue dye for purposes of identification, was injected into the sinuses of several dead rabbits. Subsequently, the sinuses were opened and were found to have retained the agar. The volume of the sinal cavity was found to be approximately 0.75 cc.

A previous experiment indicated that the agar had no appreciable effect in either impeding or promoting sensitization. Two rabbits were given subcutaneous injections of the egg white–agar combination. The resulting sensitization was then compared with that secured by the injection of egg white alone into the subcutaneous tissues. In both cases, the Arthus phenomenon was noted on cutaneous testing after five injections.

After these preliminary observations, intrasinal injections into the intact animal were made through the perforation of the maxillary sinal wall. After the hair in this area had been removed, the region of the perforation could be palpated. Above and below the fenestra were ridgelike projections of the maxillary bone. Alcohol was applied, and a 23 gage needle was introduced through the skin and the membrane sealing the perforation. The aqueous solution of egg white was combined, in these sinal injections, with agar in equal proportions. This material was easily injected by means of a syringe. Doses of 1.5 to 2 cc. were administered at weekly intervals. This quantity, at least twice the volume of the cavity, was used to insure optimum filling.

A test for hypersensitivity was made in the mucous membrane of the upper lip on each experimental animal, usually after the third and after the fifth sensitization injection. The tests were made with 0.10 to 0.15 cc. of antigen, injected intramucosally. Attempts were also made to test the mucous membrane of the sinus, but the results were inconsistent, probably because of technical difficulties, and therefore testing of this site was abandoned. According to the experiments to date, in order to perform a satisfactory test the antigen must be injected into the sinus membrane and not merely applied to it. The sinus membrane is so thin and taut as to make very difficult the insertion of a needle.

In those rabbits which were sensitized through the sinus, tests of the abdominal skin were made simultaneously with those of the mucous membrane. These were done after five sensitization injections, 0.10 to 0.15 cc. of antigen being used.

Normal rabbits were used to control all steps of the experiments.

Nine rabbits received intrasinal injections, 3 intravenous, 1 each intraperitoneal and intracutaneous and 4 subcutaneous.

RESULTS

All the animals showed hypersensitivity of the mucous membrane. After three sensitizing injections, the test dose produced similar bleblike areas measuring about 1 cm. in diameter. Some of these were bright red; others were only somewhat redder than the surrounding mucous membrane. Many revealed small yellow necrotic foci. In the highly sensitized animals there was concomitant massive swelling of the tissues of the lip in the region of the test site. The control animals showed negative reactions to the tests.

All skin tests were strongly positive. Of 7 rabbits sensitized by intrasinal injections on which skin tests were made, the Arthus reaction developed in 5.

COMMENT

The application of these experimental findings to the human being is undeterminable. Animals of different species differ widely in their reactions to sensitization. There are, however, certain implications which are noteworthy.

In the first place, the mucous membrane of the upper respiratory tract may be affected by antigens introduced into remote portals of entry. Conversely, antigens in the mucous membranes of the sinuses may influence other areas, adjacent or distant, such as the skin of the abdomen or the aural mucous membrane. This indicates that foreign proteins repeatedly introduced into the blood stream by any portal may effectively sensitize the mucous membrane. Furthermore, an antigen repeatedly absorbed through the mucous membrane can sensitize distant regions or other mucous membranes. This may conceivably be the case, for example, with regard to the lungs of patients with asthma or bronchiectasis.

Such relationships are to be anticipated, in the light of Opie's [2] work. This author showed that the antigen enters the blood stream of the rabbit in the earlier stages of sensitization by the subcutaneous route. Therefore, all parts of the body come in contact with the antigen.

It is probable that the mucous membranes of the sinuses themselves became sensitized in the animals which were given intrasinal injections. In several rabbits a nasal discharge developed, increasing with each successive injection, on the side corresponding to the injected sinus. In others marked edema was seen in the soft tissues over the sinus the day following repeated injections. Histologic examination of the sinus membranes of these rabbits disclosed edema and marked increase in the numbers of all inflammatory cells, particularly eosinophils.

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2. Opie, E. L.: The Relation of Antigen to Antibody in the Circulating Blood, J. Immunol. **8**:55, 1923.

CONCLUSIONS

1. A mucous membrane can be sensitized via many portals, including other mucous membranes.

2. Foreign substances introduced into the nasal sinus, without injury to the mucous membrane, sensitize distant areas, such as the skin and other mucous membranes.

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Case Reports

RADICAL MASTOIDECTOMY WITH COMPLETE RECOVERY IN A CASE OF CHRONIC LYMPHATIC LEUKEMIA

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In most cases of leukemia a serious infection is usually a terminating episode. This is especially true in lymphatic and monocytic leukemia, in which there is a diminished number of phagocytic polymorphonuclears, both in the blood and in the marrow. The case under consideration is interesting for several reasons. First, the patient was known to have leukemia for at least five years prior to operation; second, his blood picture showed no ultimate change due to the infection, and, third, after a radical mastoidectomy he has remained comparatively well for four years. An abstract of his case history follows:

REPORT OF A CASE

D. S., a white man 45 years of age, was admitted to Lenox Hill Hospital on Dec. 3, 1937, complaining of pain, stiffness and swelling posteriorly on the right side of his neck. The pain radiated anteriorly and up to the right temporal area and was becoming more intense. The hearing was poor in the right ear and good in the left. He had been having discharges from the right ear for many years and had similar episodes of pain five, ten, fifteen and twenty years before, though not so severe. Five years before admission he was told by an otolaryngologist that he needed an operation, but his white blood cell count was found to be so high that an operation was considered unwise by the surgeon. His white cell count at that time was said to be 45,000.

On examination the patient was apparently in severe pain and looked acutely ill. His temperature was 104 F. The left ear was normal; the right ear had a large chronic perforation, with discharge and granulation tissue in the middle ear. The cervical nodes were enlarged and tender; this tenderness spread over the back of the neck and behind the mastoid. The heart and the lungs were normal. Examination of the abdomen showed the liver enlarged to the left of the epigastrium and the spleen palpable one and one-half fingers below the costal margin, not tender, but firm. An appendectomy scar was present. The patient's temperature on admission was 104 F. and it rose to 105 that night. The urine showed albumin (1 plus), with a few hyaline casts.

After roentgen examination of the mastoids, Dr. Frank Huber reported as follows: "Left side: normal. Right side: no cellular development can be seen, and there is complete sclerosis of the mastoid region. A circular defect approximately 5/8 inch (1.6 cm.) in diameter is seen just anterior to the middle of the lateral sinus and apparently in close relation to its anterior wall. This defect probably represents a cholesteatoma."

The diagnosis was acute exacerbation of chronic mastoiditis with cholesteatoma. Examination of the blood showed the hemoglobin content 82 per cent, the red

From the service of Dr. John D. Kernan, Lenox Hill Hospital.

blood cells 4,700,000 and the white blood cells 150,000, with lymphocytes 96 per cent and polymorphonuclears 4 per cent. This blood picture indicated the presence of chronic lymphatic leukemia.

A 500 cc. blood transfusion was given preoperatively, and with the patient under anesthesia induced with nitrous oxide and ether a radical mastoidectomy was performed on the right side.

The following report of conditions seen at operation was made: "A sclerotic mastoid is observed, with a cortex not as thick as is usually found. There are a large cavity with pus under tension and a large cholesteatoma floating in the pus. The erosion involves a small portion of the sinus plate. The dural plate is intact. The cholesteatoma extends through the antrum to the middle ear. The ossicles are eroded. The sinus, when exposed, is found to have a thick fibrous wall but no pus around it."

The usual radical mastoidectomy was performed, but the plastic flap of the external canal was not made at that time, and the wound was left wide open.

Blood Counts in Case of Chronic Lymphatic Leukemia

Date	Hemoglobin, per Cent	Red Blood Cells	White Blood Cells	Lymphocytes, per Cent	Polymorphonuclears, per Cent	Eosinophils, per Cent
12/3	82	4,700,000	150,000	96	4	
			Transfusion; Operation			
12/5	100	4,900,000	180,000	97	3	
12/9	81	3,800,000	240,000	94	2	
12/11	83	3,900,000	220,000	98	2	
			Transfusion			
12/13	94	4,700,000	143,000	95	5	
12/21	80	3,500,000	92,000	98	2	
			Transfusion			
12/25	84	4,200,000	96,000	98	2	
12/28	80	4,000,000	90,000	98	2	

There was considerable bleeding during the operation. The patient underwent the anesthesia well. Culture of material from the mastoid showed hemolytic streptococcus; the blood culture was negative. Microscopic examination of the curettings showed cholesteatoma. The postoperative course was uneventful. The patient's temperature gradually came down; the wound granulated slowly.

A transfusion of 500 cc. was given seven days postoperatively and repeated twenty-one days postoperatively. The blood count is shown in the accompanying table.

Two weeks after the original operation a T flap plastic procedure was performed on the right external canal and the wound closed. The patient continued to improve, and he was discharged from the hospital on Jan. 16, 1938. The mastoid cavity epithelized slowly, and about three months after the patient's discharge from the hospital the cavity was completely healed.

The patient has been carefully followed to the time of writing, four years after operation, and has felt well. The hemoglobin content of the blood remains about 80 per cent; the white blood cell count is around 100,000, with 95 per cent lymphocytes. The sternal marrow shows marked infiltration with lymphoid ele-

ments, many of which are lymphoblasts. The spleen remains large and hard. He has had no complaints referable to the ear and no further infection. He has had no roentgen treatment at any time.

SUMMARY

A case of chronic lymphatic leukemia is presented, in which a radical mastoidectomy was performed, with complete recovery. Adequate preoperative and postoperative transfusion therapy probably contributed to the successful outcome.

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19 East Eightieth Street.

CHORDOMA

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A rare tumor, first correctly identified by Müller,[1] in 1858, is chordoma. Boldrey and McNally [2] found only 85 reported cases as of March 1941. Rea [3] quoted Cushing [4] as stating that chordoma occurs in the ratio of 1 to 1,800 among the tumors found in the region of the optic chiasm.

Chordoma develops from remnants of the notochord at the junction of the dorsum sellae turcicae and the occipital bone. It may extend into the brain stem or forward into the nasopharynx. Boldrey and McNally [2] stated that 20 per cent of tumors diagnosed as chordoma are of the nasopharyngeal type and 80 per cent intracranial. In the case to be reported in this paper the tumor was of the latter type.

In describing the notochord Piersol [5] stated:

"Coincidently with the formation of the medullary groove the entoblast opposite the bottom of that furrow exhibits proliferation and thickening; the group of cells thus differentiated becomes separated from the general mass of the inner layer and takes up a position immediately below the neutral tube. This isolated column of entoblastic cells constitutes the notochord, or the chorda dorsalis, the earliest suggestion of the cardial vertebrate axis around which the parts of the early embryo are symmetrically arranged. While for a time constituting the sole longitudinal axis of the embryo, extending from a point near the cephalic pole, which corresponds later to the base of the skull, to the caudal extremity, the notochord is but a temporary structure, and subsequently is supplanted by the true vertebral column. It is interesting to note that in the connecting link between the vertebrates and the invertebrates, the Amphioxus, the notochord remains as the permanent and sole spinal axis.

"The history of the notochord in man and mammals presents three stages: (a) it exists as an unbroken cord which extends uninterruptedly through the series of cartilaginous vertebrae; (b) the notochord suffers segmentation in such manner that the breaks in its continuity correspond to the vertebral bodies, conspicuous proliferation and local increase in its substance, on the contrary, marking the position of the intervertebral disks in which the chordal tissue during the first months after the birth is represented by a considerable mass of central spongy

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2. Boldrey, E., and McNally, W. J.: Chordoma of Basiocciput and Basisphenoid: Report of Four Cases, Arch. Otolaryng. **33**:391 (March) 1941.

3. Rea, R. L.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938.

4. Cushing, H.: Chiasmal Syndrome of Primary Optic Atrophy and Bitemporal Field Defects in Adults with Normal Sella Turcica, Arch. Ophth. **3**:704-735 (June) 1930.

5. Piersol, G. A.: Human Anatomy, ed. 5, Philadelphia, J. B. Lippincott Company, 1916.

substance; (c) atrophy of the remains of the notochord, resulting in the entire disappearance of the chordal tissue within the vertebrae substance existing within the intervertebral disks.

"The cephalic end of the notochord in man corresponds in position to the dorsum sellae, and marks the division of the skull into two parts, that lying in front of the termination of the notochord, the prechordal portion, and that containing the notochord, the chordal portion; the latter is sometimes described as the vertebral segment of the skull."

REPORT OF A CASE

A white woman aged 66 was seen in October 1936. She complained of itching and burning of her eyes and transient blurring of her vision. Except for conjunctival irritation the eyes were normal externally. Tension, as recorded by the Schiötz tonometer, was within normal limits. The patient's glasses required no change. Vision was 20/16 in each eye. Muscular balance was normal. The fundi were normal. A general physical examination revealed mild hypertension. A dental examination, including roentgenograms, showed a few abscessed teeth, which were removed later. The conjunctival infection was relieved by simple treatment.

Eleven months later the patient returned, complaining of tearing, pain in the right eye and blurring of vision, which had been noted occasionally for several weeks. She also complained of blockage of the ears. An examination of the ears, nose and throat gave negative results except for findings as seen in chronic catarrhal otitis media. No audiograms were made. The conjunctiva was congested; zinc sulfate drops were prescribed. An examination showed the vision to be 20/16 in each eye; tension, muscular balance and fundi were normal. The right eye, including the fundus, remained normal throughout the illness. The following day the patient suddenly lost the vision in her left eye for two hours, after which it slowly became normal. The fields of vision were irregularly contracted. No scotomas were found. Blurring of vision continued, and on the sixth day the vision in the left eye was reduced to 20/200. On the seventh day the vision in the left eye was barely perception of light. The patient had been given a vasodilator drug for several days, but administration was discontinued at this time.

The patient was seen by a consultant, who was unable to diagnose the condition. He advised a roentgen examination of the sinuses and the tamponing of each naris with a solution of a silver salt and epinephrine.

On the eighth day the pupillary reflex of the left eye was absent except for the consensual reflex. Proptosis was present. The cranial nerves reacted normally.

The roentgen report was as follows: "The frontal and ethmoid sinuses and the antrums are clear. The sphenoid region is cloudy. There is apparently destruction of the posterior bony wall of the sphenoid sinus and also destruction of the sphenoid bone into the floor of the cranium. The posterior clinoid of the sella turcica is also completely destroyed. Conclusion: There is probably a neoplasm springing from the posterior part of the sphenoid and extending through the floor of the skull and the posterior wall of the sphenoid sinus."

The patient was sent immediately to a neurologic surgeon, who concurred with the diagnosis made with the aid of the roentgenograms. On the sixteenth day he attempted to remove the growth by the intracranial route but was unsuccessful in locating the mass. The operation was interrupted when the patient was over-

come by shock. Just before the operation, the patient had been unable to speak, and hemiplegia of the right side had been noted. She did not regain consciousness after the operation. Two days after the operation she died.

At autopsy the tumor mass was located at the sella turcica.

The gross specimen was described as follows: "The specimen is a mass of blood clot, roughly 2 by 3 by 3 cm., which contains irregular bands and masses of soft yellowish tissue which grossly has the appearance of tumor. In cutting through the mass to obtain blocks for section, there was found in its central portion an irregular mass of yellowish tissue, roughly 0.5 cm. in diameter, which was much firmer in consistency than the surrounding tissue."

A diagnosis of intracranial chordoblastoma was made from the microscopic section.

COMMENT

A fetal condition which should be transitory but is retained permanently is called by Arey,[6] "developmental arrest." Arey [6] expressed the belief that such a defect is due to improper timing in the development of the various structures of the body. This, in turn, is caused by faulty physiologic chemistry, or metabolic disturbances, within the fetus. Timing is of utmost importance.

High voltage roentgen therapy prior to the operation might have been of value.

The onset and the rapid development of the symptoms are facts of interest in this case.

700 Church Street.

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6. Arey, L. B.: Developmental Anatomy, ed. 3, Philadelphia, W. B. Saunders Company, 1934.

ADAMANTINOMA OF THE FLOOR OF THE RIGHT NOSTRIL

Occurrence in a Child Aged Five Years

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The difficulty in recognition of the presence of a slow-growing malignant neoplasm involving the floor of the right nostril in a child aged 5 years prompts the report of this case. At the first observation the lesion had the picture of a furuncle involving the nasal floor.

The adamantinoma is a slow-growing tumor, insidious in onset. The symptoms may be so slight that not until the tumor has advanced can it be recognized clinically. Roentgenograms of the teeth and sinuses are inconclusive. It is a tumor that destroys by invasion and secondary infection. It is resistant to roentgen radiation but does respond to a degree, as this case demonstrates.

The adamantinoma arises from cells concerned with tooth development and is classified under the general heading of epithelial odontomas. It is multilocular and microscopically shows masses of epithelial cells of the enamel organ type of ameloblasts, cuboidal cells and stellate reticulum. At an early stage the tumor may be solid rather than cystic. It has been called an epithelioma by many investigators, notably Bloodgood.[1] On the other hand, Waldron stated that it should not be so considered.

Bloodgood, in a report of 32 cases recorded at the Johns Hopkins Hospital from 1889 to 1930, showed that when the tumor remained in the bone shell and was completely removed the patient was cured but that this did not occur often. He cited the case of a man who died twenty-nine years after his first operation of involvement of the brain. The patient had twelve operations during this period. He cited another case in which the tumor was first observed at the age of 13 and had a duration of six years. During this period there were eight operations, with rapid recurrence after each. The ninth operation resulted in death.

In my opinion, the clinical course and the gross and microscopic appearance of the adamantinoma justifies one in considering it a slow-growing malignant epithelioma. It is made up of epithelial elements, invades normal tissue, recurs and destroys when it involves vital structures.

The prognosis depends on the position and extent of the tumor, as Waldron [2] and Öhngren [3] pointed out. Tumors arising in the mandible

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From Children's Hospital of the East Bay.

1. Bloodgood, J. C.: Lesions of the Oral Cavity and of the Jaws and the Region of the Jaws, in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1930, vol. 4, chap. 4, p. 265.

2. Waldron, C. W.: Tumors of the Upper Jaw, Surg., Gynec. & Obst. **72:** 511, 1941.

3. Öhngren, L. G.: Malignant Tumors of the Maxillo-Ethmoidal Region, Acta oto-laryng., 1933, supp. 19, p. 1.

and the maxilla are more favorable for cure than those arising higher and invading the deeper structures.

Successful treatment consists in complete surgical eradication of the tumor, followed by high voltage roentgen irradiation. The patient should be under constant supervision so that a recurrence may be noted and the growth removed.

REPORT OF A CASE

Present Illness.—On Nov. 24, 1939, C. L. W., a girl aged 5 years, was referred to me by her pediatrician, Dr. Harry Horwitz, because she had a swelling in the floor of the right nostril which was suppurating. A diagnosis of furuncle was made and roentgen therapy advised. This was carried out, with complete disappearance of the lesion after three treatments.

On Aug. 1, 1940 a mass was again noted and was much more extensive, pushing forward the right ala nasi and the right portion of the upper lip and involving the nasal floor. The diagnosis of a malignant growth was considered and biopsy recommended. The mother wished to try high voltage roentgen ray therapy, and several treatments were given. The mass again disappeared. On Nov. 11, 1940 the child returned with a growth larger than before. The tumor now seemed to involve the maxilla under the right ala nasi and extended inward to include the lateral wall and the floor of the nasal vestibule and the anterior portion of the inferior turbinate. The roentgenographic report was: "There is no evidence of involvement of the maxilla."

Past and Family History.—The child had been singularly free of disease except for the present illness. Her tonsils and adenoids were removed in May 1940, with an uneventful recovery.

The father, aged 35, had an epithelioma of the tongue; the mother and a sister were living and well.

Physical Examination.—On admission to the hospital the child appeared ill. She was somewhat emaciated, and was shorter than is usual for her age. The right side of the upper jaw was swollen, and the right nostril and the right side of the upper lip were pushed forward. On palpation a firm mass was outlined, involving the area under the right nostril and the alveolar process and extending into the nose. The nasal floor and the lateral wall of the vestibule were invaded as well as the anterior end of the inferior turbinate.

Laboratory Examination.—Studies of the blood revealed 11.6 Gm. of hemoglobin per hundred cubic centimeters (68.5 per cent) and 7,300 white cells per cubic millimeter. Of these, eosinophils constituted 4 per cent, neutrophils 49 per cent, with 3 per cent stab and 46 per cent segmented forms, leukocytes 37 per cent and monocytes 10 per cent. The bleeding time was three and a half minutes and the coagulation time (capillary) three and a half minutes.

Gross and microscopic examination of the urine gave normal results.

Operation (March 9, 1941).—With the patient under general anesthesia, a sublabial incision was made along the entire upper alveolar margin, and the skin and mucous membrane were elevated, revealing the tumor. The neoplasm had its origin in the area above the right lateral incisor and involved the floor of the right nasal vestibule and the inferior turbinate. A dense fibrous capsule surrounded the mass and could easily be separated from the surrounding tissue. Another mass, just posterior, involved the floor of the nose and the inferior tur-

binate. This was removed, with a margin of what appeared to be normal tissue. The anterior wall of the maxilla was normal in appearance and was not included in the procedure.

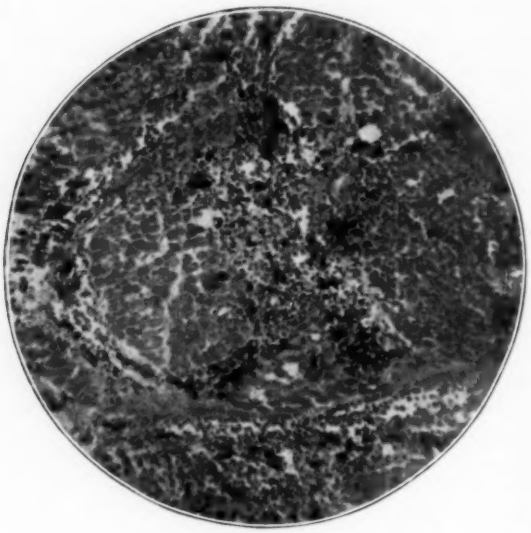

Fig. 1.—Section from the first mass removed, showing cell infiltration and chronic inflammatory reaction.

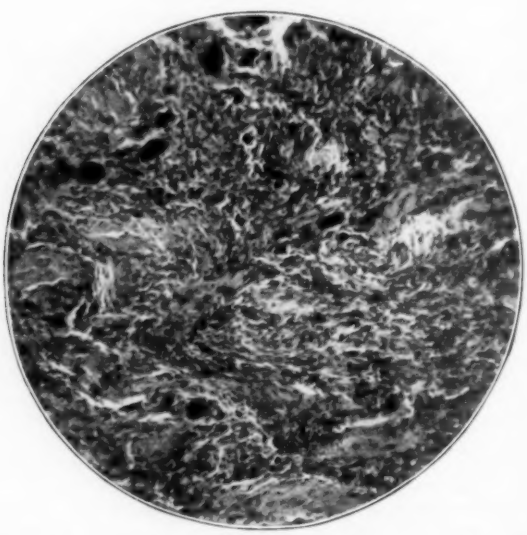

Fig. 2.—Section showing dense fibrous stroma and spindle-shaped cells.

Pathologic Examination.—The specimen submitted consisted of two rather fibrous masses of tissue, one measuring 3.5 by 2 cm. and the other 1.5 by 1 cm. They were rather discrete and elastic.

Sections showed an invading tumor composed of round, oval and spindle-shaped cells, with a moderate number of mitotic figures. In some places there was a pseudopapillary structure. The tumor had a partial fibrous capsule and a dense fibrous stroma.

The diagnosis (Dr. Paul Michael) was adamantinoma.

Postoperative Course.—The operative wound healed per primam in three weeks. The child was then given high voltage roentgen radiation to the area in an amount consistent with her age. She has gained about 10 pounds (4.5 Kg.) in weight and, according to her mother, is much more active than ever before. The nasal floor has healed, as has the lateral wall, and appears normal.

Prognosis.—Time alone will tell whether a cure has been obtained. The cases reported by Bloodgood are not encouraging.

SUMMARY

A case of adamantinoma in a child aged 5 years is reported. This tumor is characterized by its slow growth and its recurrence after high voltage roentgen irradiation. A point of interest in this case is that the father had an epithelioma of the tongue.

508 Sixteenth Street.

CHANCRE OF THE NASAL SEPTUM

ARTHUR J. CRACOVANER, M.D., NEW YORK

Extragenital primary syphilitic lesions are not uncommon. Otolaryngologists occasionally encounter a chancre on the nose, but seldom does one see a primary lesion inside the nose. In the literature one finds a number of references to external nasal lesions but very few to internal chancre of the nose. Because of the rarity of this lesion and because it is important to bear the possibility in mind, I feel that it is worth while to report this case.

External primary lesions are usually due to faciogenital connection but may also be due to finger contamination with the organism. Internal lesions are probably due entirely to finger contamination and are more likely to be found in obstetricians, gynecologists, nurses and midwives. The case to be presented is therefore important also in that it reminds physicians and nurses to be more careful in protecting themselves from infection.

Chancre within the nose is difficult to diagnose unless one keeps the possibility in mind. It appears as a hard indurated lesion and increases rapidly in size. There is an acute inflammatory reaction around it. It causes nasal obstruction and therefore difficulty in breathing. It must be differentiated from a tuberculous lesion, gumma and carcinoma. These are not frequently accompanied with the acute inflammatory reaction which usually surrounds a chancre. A careful history, a dark field examination, if done early, and a Wassermann or Kline test will aid in making the diagnosis.

REPORT OF A CASE

C. Z., a white woman aged 27, a registered nurse, was admitted to Lenox Hill Hospital on Aug. 2, 1939.

She had had pain and nasal obstruction for about six weeks. She awoke each morning with a feeling of soreness in her nose. Three weeks before admission a head cold developed and nasal and postnasal discharge, which failed to subside in the usual time. She lost her sense of smell, and glands in her neck became swollen. One week before admission the patient noticed that she had some "sores" in her nose, on both sides. The "sores" had crusts on them. They were dry, and there was no bleeding. Her temperature two days before admission was 101 F. The rest of the history at that time was irrelevant.

Examination revealed marked obstruction of the left naris. This seemed to be due to a thickening of the septum. Anteriorly over the cartilaginous portion there was a raised crater-like ulceration, which appeared hard and indurated and was surrounded by an inflammatory reaction. It was covered with a gray exudate. The thickening of the septum caused partial obstruction of the right naris, but there was no ulceration on this side. The mucous membrane could not be shrunk enough to permit one to see beyond the area of ulceration.

From the service of Dr. John Kernan, Lenox Hill Hospital.

Examination of the neck showed enlargement of the anterior and posterior cervical nodes, more marked on the left side. They were not tender and were quite discrete.

The possibility of abscess, tuberculosis, gumma and carcinoma occurred to me. Aspiration of the septum gave negative results. Roentgen examination of the chest showed a small area of calcification at the base of the right lung and at the apex of the left lung and several more at both hilar regions, which represented completely healed tuberculous foci. Roentgen examination of the sinuses showed a slightly thickened mucous membrane throughout. Biopsy showed granulation tissue with considerable fibroblastic proliferation and a dense infiltration with numerous round cells. A few plasma cells and occasionally polymorphonuclear leukocytes were seen. There were no giant cells or caseation necrosis.

Examination of the blood showed the hemoglobin content 76 per cent, red cells 4,000,000 and leukocytes 7,700, with polymorphonuclears 62 per cent, lymphocytes 32 per cent, monocytes 3 per cent and eosinophils 3 per cent.

The Wassermann and the Kline reaction were reported to be 4 plus. A more definite history was taken then, and it was found that three months before admission the patient had been working at another hospital and was assigned to the nursery. Before she accepted this assignment, a Wassermann test was found to be negative. While in the nursery she was taking care of children with infected umbilical cords. She remembered that she took entire care of a Negro child who had a syphilitic infection of the cord. The mother of the child had a lesion which was probably a chancre, though the physicians at the time were in disagreement.

At this time a rash developed on the patient, which was diagnosed definitely by the dermatologist, Dr. Leo Speigel, as a secondary rash. He agreed then that the original infection had probably been contracted about two months previously and that the lesion in the nose was a primary lesion. Subsequently, under the accepted treatment for syphilis, the patient was cured of her disease.

Besides the importance of the diagnosis in a case of this kind, the question of compensation also arises. This patient, a trained nurse, contracted her disease while on duty in the hospital. She refused to claim compensation because of possible embarrassment.

Since the lesion is so easily confused with others and may even be unrecognized, proper treatment, which should be started immediately, may be delayed. It is hoped that this report will serve to bring the possibility of primary syphilitic internal nasal lesion once again to the minds of otolaryngologists.

103 East Seventy-Eighth Street.

Clinical Notes; New Instruments and Technics

TONSIL KNIFE AND ADENOTOME FOR USE WITH HEAD IN THE HYPEREXTENDED POSITION

GEORGE E. SHAMBAUGH JR., M.D., CHICAGO

Removal of tonsils and adenoids with the head in the hyperextended position is favored by many otolaryngologists because it eliminates all possibility of aspirating blood and detritus from the tonsils, with the hazard of postoperative pulmonary infection (pneumonia and abscess). The special curved tonsil knife [1] (fig. 1) has been found advantageous in dissecting the tonsil free from the pillars, with preservation of a maximum of mucous membrane on the pillars. The short straight blade, at one end, is used to make the initial incision through the plica triangularis at the lower pole, just behind the anterior pillar. The curved blade, at the other end, is then inserted into the incision between the layers of the capsule of the tonsil and is drawn upward along the border of the anterior pillar to the upper pole, using the cutting edge on the *concave* side of the curve. Without removing the curved blade from the incision, the blade is now carried down along the edge of the posterior pillar, using the cutting edge on the *convex* side of the curve. The tip of the curved blade is blunt, to prevent it from piercing through the capsule either into the tonsillar or into the muscle tissue.

When the head is in the hyperextended position and the Davis-Crowe mouth gag is used difficulty is sometimes experienced in removing with the usual La Force adenotome (fig. 2*A*) the adenoid tissue that lies forward, close to the posterior end of the septum. To obviate this difficulty an adenotome [1] has been designed with a reversed curve (fig. 2*B*) for use with the head in the hyperextended position.

122 South Michigan Boulevard.

1. Instruments are supplied by the American Optical Company, 10 South Wabash Avenue, Chicago.

Fig. 1.—Tonsil knife. At one end is a straight blade and at the other a curved blade with a blunt point and both edges sharp.

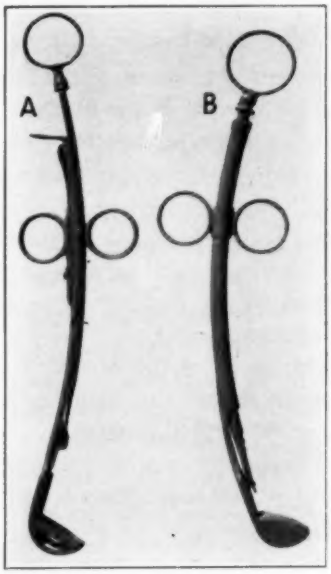

Fig. 2.—*A*, the usual La Force adenotome; *B*, a special curved adenotome for use with the head in the hyperextended position.

Progress in Otolaryngology

Summaries of the American and British Bibliographic Material Available in the Field of Otolaryngology

PERORAL ENDOSCOPY

LOUIS H. CLERF, M.D.
AND
F. JOHNSON PUTNEY, M.D.
PHILADELPHIA

THE RESPIRATORY TRACT

Anatomic and Physiologic Considerations.—The diameters of normal and abnormal bronchi were determined by Chapman and Smith [1] by the use of bronchograms. On the right side the mean diameter of normal bronchi was 4.76 mm. and of abnormal bronchi 5.33 mm. On the left side the mean diameter of normal bronchi was 4.84 mm. and of abnormal 5.61 mm. No significance was attached to the findings of slightly larger bronchi on the left side and slightly larger bronchi in men than in women. Likewise there was no significant relation found between bronchial diameter and body weight and between bronchial width and the size of the chest.

Functional-emotional spasm as a cause for disturbance in swallowing has been suggested by Faulkner,[2] and he also has noticed that emotional factors produce bronchial spasm or relaxation. One case was reported in detail in which thoughts of insecurity and frustration brought about bronchial spasm and narrowing of the bronchial lumen and suggestions of a pleasant and desirable nature caused bronchial relaxation. Within fairly wide limits and with similar groups of people the author would expect suggestions and proposals to produce similar bronchial responses, which may prove useful in the treatment of bronchial spasm and associated asthmatic conditions.

In an investigation of the action and influence of expectorants and gases on the secretions of the tracheobronchial tree, Holinger, Basch and Poncher [3] carried out studies to evaluate their actions, to secure

1. Chapman, J., and Smith, H. E.: Diameters of Normal and Abnormal Bronchi: Statistical Study, Am. Rev. Tuberc. **43**:504 (April) 1941.

2. Faulkner, W. B.: Influence of Suggestion on Size of Bronchial Lumen, Northwest Med. **40**:367 (Oct.) 1941.

3. Holinger, P.; Basch, F. P., and Poncher, H. G.: The Influence of Expectorants and Gases on Sputum and the Mucous Membranes of the Tracheobronchial Tree, J. A. M. A. **117**:675 (Aug. 30) 1941.

data regarding their use and also to ascertain if other methods might be employed to secure similar or preferably more beneficial results. Two factors responsible for liquefying sputum in the bronchi consist of agents which increase hyperemia of the mucosa as well as agents which increase the rate and depth of respiration. While the expectorant drugs almost uniformly aid in liquefying a portion of the secretions which lie in the larger bronchi, they rarely affect the secretions in the more dependent and peripheral bronchi.

The actions of gas inhalations are quite specific. They grossly influence the physical and chemical qualities of both expectorated and bronchoscopically removed sputum, as well as the character of the bronchial mucosa. The inhalation of steam or of a highly humidified atmosphere results in liquefaction of sputum. Carbon dioxide has an action similar to that of steam but more intense. In addition, it increases the resorbing power of the bronchial mucosa and may be considered as an extremely efficient expectorant. Oxygen acts as an antiexpectorant, and therefore its use alone is contraindicated in the presence of obstructive lesions associated with large quantities of secretion. This unfavorable effect may be neutralized in part or completely by the addition of steam or of 5 to 10 per cent carbon dioxide.

The employment of codeine and atropine over long periods is contraindicated in cases of bronchopulmonary suppuration producing obstructive symptoms. Atropine, however, may be employed prior to bronchoscopy to aid in reducing the total amount of secretion. In cases of bronchiectasis and in certain types of asthma in which bronchial obstruction plays a role in the symptoms and pathologic changes, frequent inhalations of carbon dioxide by mask together with steam inhalations may be used to augment postural drainage and bronchoscopic aspiration, which are fundamentally important. The employment of steam and carbon dioxide between bronchoscopic aspirations and especially prior to bronchoscopy is indicated if the sputum is known to be thick and tenacious.

In cases of laryngotracheobronchitis the employment of a humidified atmosphere aids greatly in liquefying secretion. If obstruction to the airway progresses in spite of removal of secretions, oxygen may become necessary. While deep breathing and cough will commonly aid in spontaneously relieving patients with postoperative collapse of the lungs, steam inhalations aid in liquefying secretions, thus facilitating their removal, and inhalation of carbon dioxide will increase the rate and depth of respiration as well as aid in liquefying secretions. If these agents fail bronchoscopic aspiration should be resorted to.

Pressman [4] described the unusually wide to and fro excursions in a lateral direction of the carina which he has observed in cases of pleural effusion. The widest excursions were found in cases of massive effusions on the left side, but they were also observed in cases of effusion on the right side. He explained this as a "fluid wave" produced by the pulsating heart and conveyed to the main stem bronchi; this transmission of movement manifests itself by deviation of the carina. While the practical application of this observation may be limited, it is nevertheless of some value in some cases of small or localized effusions in which the clinical diagnosis may be uncertain.

Acute Laryngotracheobronchitis.—The anatomic peculiarities of the larynx in early life are in part responsible for the frequent stormy manifestations of otherwise mild infections of the air passages. In a report on acute laryngeal conditions in children, Heatly [5] directed attention to the difficulties in diagnosis as well as in treatment. He emphasized the necessity for careful examination of the larynx so that differential rather than inferential methods of diagnosis would be more generally applied. Reports vary widely concerning the organisms most commonly encountered in acute laryngotracheobronchitis. While the disease in a majority of the cases is caused by hemolytic streptococci and in most of these sulfanilamide proves a valuable adjunct, it is of interest to call attention to bacteriologic studies made in Heatly's cases. In a series of 28 cases of nondiphtheritic laryngitis, the predominant organism proved to be a green-producing streptococcus. He emphasized the importance of not relying too strongly on chemotherapy alone. Early tracheotomy with frequent aspiration of the viscid secretions from the tracheobronchial tree is of paramount importance. He also agreed that the employment of physiologic solution of sodium chloride or a dilute solution of sodium bicarbonate instilled through the tracheotomic cannula stimulates the cough reflex and aids materially in the effectiveness of suction treatment.

While the cause of acute laryngotracheobronchitis has not been definitely determined and many organisms have been obtained from the laryngeal and tracheal secretions which may be considered as causative factors, it has been recently suggested that a virus is the primary invader which prepares the way for secondary bacterial invasion, especially by hemolytic streptococci. In a review of this subject and in a report of 62 cases with postmortem studies in 8, Orton and his associates [6] dis-

4. Pressman, J. J.: A Bronchoscopic Observation in Cases of Pleural Effusion, Arch. Otolaryng. **34**:844 (Oct.) 1941.

5. Heatly, C. A.: The Management of Clinical Problems Involving the Larynx in Infancy and Childhood, New York State J. Med. **41**:607 (March 15) 1941.

6. Orton, H. B.; Smith, E. L.; Bell, H. O., and Ford, R. A.: Acute Laryngotracheobronchitis, Arch. Otolaryng. **33**:926 (June) 1941.

cussed etiologic factors, pathologic findings, diagnostic procedures, symptoms and treatment. A number of illustrative case histories are cited to emphasize certain of these phases. A detailed report of the autopsy observations is included in their report.

They concluded that acute laryngotracheobronchitis in young children is an important problem both for the general practitioner and for the pediatrician. Proper handling of this disease requires early recognition. When signs of obstructive dyspnea appear, valuable time should not be lost in treating the condition with diphtheria antitoxin. When acute laryngotracheobronchitis is suspected the patient should be transferred immediately to a hospital where direct laryngoscopy and bronchoscopy can be carried out at once and where more active treatment, such as intubation and tracheotomy, may be done at a moment's notice, with a trained personnel always immediately available. If time permits roentgen study should be made prior to operation; otherwise it should be done immediately after tracheotomy. Intubation should be done only for immediate relief; this should be followed by tracheotomy. Tracheotomy is the operation par excellence. The patient should be placed in a room in which the air is supersaturated with moisture and maintained at a temperature of 70 F. at all times. This temperature with a high humidity provides a more comfortable environment for the patient than the old-fashioned hot croup tent. By the use of a mechanical humidifier the humidity in a tent can be raised to 95 per cent when the room temperature is 70 F. The temperature and humidity of an oxygen tent have more effect on the comfort of the average patient than does the concentration of oxygen. If the temperature of the air is not above 70 F. loss of water from the body can be decreased by increasing the humidity.

The problems in diagnosis as well as in treatment of acute laryngotracheobronchitis are due to the formation of a thick membrane-like exudate as well as marked swelling of the mucosa and submucosa of the larynx, trachea and bronchi and the development of severe dyspnea. In discussing this subject, LeJeune and Bayon [7] emphasized the importance of humidification to aid in getting rid of thick secretions. If dyspnea becomes a prominent symptom tracheotomy should be resorted to promptly. They expressed the opinion that tracheotomy is best done by first inserting a bronchoscope into the trachea and then doing an orderly operation. This not only provides a satisfactory airway but, since a low tracheotomy can be performed, facilitates the removal of secretions by the aid of aspiration through the cannula. Frequent aspirations may become necessary, and for this reason it is important

7. LeJeune, F. E., and Bayon, P. J.: Acute Laryngotracheobronchitis, J. A. M. A. **117**:1316 (Oct. 18) 1941.

to have available the service of a nurse experienced in the care of tracheotomized patients. The solutions commonly employed for thinning secretions are not always successful. These authors have employed a preparation of papaya (essence of caroid), and in one of their cases it appeared to exert an excellent effect. While the sulfanilamide compounds should be of value, there have been insufficient data collected to permit of conclusions. In a few of their cases in which a heavy growth of staphylococci was secured they employed a spray of the sodium salt of sulfathiazole.

In a report of 14 cases of acute laryngotracheobronchitis in which it was necessary to perform tracheotomy, Cummings [8] reported a mortality rate of 50 per cent. While hemolytic streptococci were the organisms most frequently found, any of the common bacteria except the diphtheria bacillus may be recovered in pure or mixed cultures. The viscid secretions and the progression downward of the disease into the smaller bronchi tend to produce occlusion of the small airway by secretions, crusts and edema. Cummings recommended rest in bed, highly humidified air, adequate intake of fluids, tracheotomy or intubation, instillation into the tracheal cannula of physiologic solution of sodium chloride to prevent drying of secretions and bronchoscopic removal of crusts and secretions. If a staphylococcus is present bacteriophage is recommended. In addition, he believed that sulfanilamide compounds should be given. A brief summary of the 14 cases is included in the article.

The close resemblance of the clinical symptoms of acute laryngotracheobronchitis to diphtheria and pneumonia accounts for much of the confusion which is noted in the diagnosis and early treatment of this condition. In discussing this, Connell and Trowbridge [9] agreed that tracheotomy should be performed without delay for relief of the highly obstructive dyspnea. Bronchoscopic treatment and intubation have been employed to secure relief, but the results have not been encouraging. The nature of the infection precludes the use of intubation, for this method does not afford a means by which thick secretions and plugs can be frequently removed. Likewise, bronchoscopic removal of secretions further exhausts an already worn-out patient, and the time consumed in the procedure makes the method impractical. Occasionally, however, bronchoscopic removal of secretions through the tracheotomy opening may become necessary when aspiration by catheter through the tracheal cannula is unsuccessful. They recommend the prompt administration of

8. Cummings, G. O.: Acute Laryngotracheobronchitis with Reports of Fourteen Cases Which Came to Tracheotomy, Laryngoscope **51**:382 (April) 1941.

9. Connell, E. S., and Trowbridge, B. C.: Acute Streptococcic Laryngotracheobronchitis, Arch. Otolaryng. **33**:717 (May) 1941.

sulfanilamide or one of its derivatives and constant supportive care to aid in bolstering up the patient's defense mechanism. Eight cases with 3 deaths are recorded.

In a comprehensive review of the literature on laryngotracheobronchitis, Kayser [10] emphasized certain of the important advancements made in diagnosis and treatment. He also reported in detail the case of a patient who was successfully treated. He stated the belief that for patients with slight dyspnea intubation is a helpful and curative procedure but that when dyspnea is more severe tracheotomy seems to be the more appropriate measure. He recommended a transverse incision of the skin for tracheotomy and the use of Richard's crucial incision into the trachea.

Hagaman [11] reported 3 cases of laryngotracheobronchitis that terminated fatally. In 2 cases spontaneous pneumothorax developed, while in the third case diphtheria was found. In the third case cultures of material from the larynx and pharynx did not contain the organism, but diphtheria bacilli were grown from the pseudomembrane in the trachea and bronchi.

The term croup is used to designate a state of obstruction to the entrance of air into the lungs as a result of an inflammatory process either in the larynx alone or extending to the lower part of the respiratory tract. It is characterized by inspiratory stridor, hoarseness or aphonia and cyanosis or pallor, accompanied by suprasternal, epigastric and intercostal retraction. There are two main types, diphtheritic and nondiphtheritic.

Gilbert, Meyersburg and Silverberg [12] investigated a series of 226 cases and observed that while there has been a tremendous decrease in the number of cases of diphtheritic croup, the number of cases of nondiphtheritic croup has remained about the same. A careful analysis of old hospital records revealed insufficient information for dependable evaluation of the various types of croup. They therefore submitted a more comprehensive classification of these cases and considered them as instances of diphtheritic and nondiphtheritic croup. The group of cases of nondiphtheritic croup is further subdivided into cases of acute catarrhal laryngotracheitis, supraglottic edematous obstructive laryngitis, subglottic obstructive laryngitis and tracheitis, acute obstructive laryngo-

10. Kayser, R.: Acute Laryngotracheobronchitis: Report of a Case and Review of the Literature, Arch. Otolaryng. **33**:795 (May) 1941.

11. Hagaman, V. D.: Laryngotracheobronchitis, South. M. J. **34**:1082 (Oct.) 1941.

12. Gilbert, J. G.; Meyersburg, H., and Silverberg, J. S.: Croup: Preliminary Report on One Year's Investigation of Two Hundred and Twenty-Six Cases, Arch. Otolaryng. **34**:281 (Aug.) 1941.

tracheobronchitis and unclassified forms. In this classification it is important to check the clinical observations with the minute anatomic and pathologic studies. Laryngoscopic examination was performed in all the cases of their series in which there was doubt as to the pathologic evidence, prognosis or therapy. They found it necessary to use the laryngoscope in more than 30 per cent of the cases. The incidence of morbidity and mortality was affected by the occurrence of infection with Staphylococcus aureus haemolyticus as well as by complicating bronchopneumonia, which was present in 25 per cent of cases. Subglottic edematous obstructive laryngitis and acute obstructive laryngotracheobronchitis are most frequently complicated by pneumonia. A state of exhaustion from prolonged air hunger must be carefully differentiated from so-called toxic diphtheria. Measures directed to the relief of laryngeal obstruction are not contraindicated by coexisting pneumonia.

Shuman [13] reported 3 cases of acute laryngeal obstruction as a complication of measles. This rare complication may occur at any stage of measles, and although the edema observed was chiefly supraglottic prompt treatment was necessary to relieve the obstruction. Intubation was used by the author.

Raven [14] reported a case of primary Haemophilus influenzae tracheitis. The disease was self limited and nonepidemic and ran the usual course of grippal infections. The diagnosis was made from culture of the sputum.

Two cases of staphylococcic tracheobronchitis necessitating tracheotomy were reported by Shulman.[15] Staphylococci were recovered in pure culture from the throat or from coughed-up plugs of secretion, and in 1 case a staphylococcic bacteremia was present. After the use of sulfamethyltniazole (2-[paraaminobenzenesulfonamido]-4-methylthiazole) and sulfathiazole the patients recovered.

In a study of cases of acute tracheobronchitis Grier [16] observed enlargement of the superior mediastinal shadow on roentgenograms of the chest. This suggested a persistent thymus, and the question of irradiation had to be decided. Grier pointed out that the mediastinal shadow in each of these cases was rather characteristic, being symmetric and projecting on both sides of the spine, with the edges usually concave

13. Shuman, H. H.: Acute Laryngeal Obstruction as a Complication of Measles, New England J. Med. **224**:727 (April 24) 1941.

14. Raven, C.: Tracheitis Due to Bacillus Hemophilus Influenzae, Ohio State M. J. **37**:445 (May) 1941.

15. Shulman, M. H.: Staphylococcic Tracheobronchitis Treated with Sulfamethylthiazole and Sulfathiazole: Report of Two Cases, New England J. Med. **224**:978 (June 5) 1941.

16. Grier, G. W.: Mediastinal Enlargement in Acute Laryngotracheobronchitis, Ann. Otol., Rhin. & Laryng. **50**:941 (Sept.) 1941.

although occasionally straight. In contradistinction to this, the thymic shadow is almost always bulging, with the borders convex, and it not uncommonly projects to one side of the spine only.

Bronchial Obstruction.—A clinical method for the functional examination of respiration which records the movement of air in and out of the chest by means of a modified metabolism apparatus has been described by Andrews.[17] By this method of examination one can determine: the quantitative characteristics of respiration; the rate of respiration; the ventilation or minute volume; the rate of oxygen absorption; the vital capacity, inspiratory or expiratory, and its component parts; complemental, tidal and supplemental air and the velocity of air flow during the various phases of the vital capacity, usually the maximum velocity during expiration, and the maximum ventilation—that is, the largest quantity of air the patient can breathe in one minute. A series of studies was carried out on normal subjects, and observations also were made on patients with bronchial asthma, obstructive hypertrophic pulmonary emphysema, laryngeal obstruction and tracheal obstruction. On the basis of his observations three deductions may be made: First, positive pressure applied to the airway results subjectively in easier breathing for those patients who show marked collapse of the trachea or main bronchi on expiration. Secondly, in the majority of cases a patient with a considerable quantity of thick secretion insufficiently removed by cough and ciliary action shows a variability in successive determinations of vital capacity generally of an increasingly obstructive nature; it is this kind of patient with bronchial asthma who is most consistently benefited by bronchoscopic therapy. Thirdly, a patient with laryngeal, tracheal and high bronchial obstruction shows a symmetric retardation of the rate of air flow at the extremes of the determinations of vital capacity.

Bronchial obstruction plays an extensive and variable role in pathologic changes in the lung and may exert widespread effects which are not limited to the lung distal to the obstruction. Its influence on the lung is usually determined more specifically by the location of the lesion and the degree of obstruction than by the etiology of the process which is producing the obstruction. In a discussion of this subject Holinger and Andrews[18] pointed out that gross significant changes in intrathoracic pressure, circulatory changes, extensive suppurative processes and other physiologic as well as pathologic processes result from obstruction. They discussed principally the mechanics of bronchial obstruction and the signs and symptoms which are produced. Obstruction of a bronchus

17. Andrews, A. H., Jr.: The Functional Examination of Respiration in Obstructive Diseases, Ann. Otol., Rhin. & Laryng. **50**:917 (Sept.) 1941.

18. Holinger, P., and Andrews, A. H., Jr.: Bronchial Obstruction: Signs, Symptoms and Diagnosis, Am. J. Surg. **54**:193 (Oct.) 1941.

interferes with the conduction of air to and from the lungs as well as with the drainage of secretions and all material from the lung. The final preparation of the air for the lungs is not materially affected by the presence of obstruction. In addition to the physiologic effects of obstruction on the respiratory action they exert a definite cardiovascular effect. Mild obstruction results in dyspnea which remains compensated by reflex and physiochemical stimulation, while severe obstruction results in respiratory decompensation or anoxia. Cardiovascular phenomena manifest themselves by pulmonary edema and finally a circulatory failure due to rising intrathoracic negative pressures. A list of the possible causes of obstruction is presented. These include extrabronchial as well as endobronchial causes. The importance of roentgen study, including bronchography, and bronchoscopy as diagnostic aids is discussed.

Martin [19] discussed the value of tracheobronchoscopy and esophagoscopy in diagnosis and treatment. The first published account of endoscopy in Edinburgh was by Dr. Logan Turner and Dr. J. S. Fraser in 1914. Since then the field has been widened. Martin discussed the increased scope of these procedures. He found it difficult to believe that children around the bronchoscopic clinics of Philadelphia—an area with about the same population as around Glasgow and Edinburgh—inhale more foreign bodies than children in these districts of Scotland. Martin asked: "Is it not more likely that the possibility of the presence of a foreign body is often overlooked in this country"? He emphasized the need of more bronchoscopic clinics in his country and suggested the possibility of several areas being served by a central clinic.

In discussing bronchoscopy as a diagnostic aid Clerf [20] emphasized that it still is the only method to be considered for the removal of bronchial foreign bodies. The greatest field of usefulness now for bronchoscopy is in the diagnosis of obscure bronchopulmonary disease and for the treatment of known conditions that respond favorably to bronchoscopy. Bronchoscopy is to the internist and the thoracic surgeon what cystoscopy is to the urologist and ophthalmoscopy is to the ophthalmologist. It is a method of direct examination; it has brought the tracheobronchial tree where one can visualize it directly. It has done much to discourage inferential diagnosis in pulmonary disease. It is one of the aids to diagnosis and treatment of pulmonary conditions that should be available in every general hospital. As a diagnostic aid it is indicated in all cases of pulmonary disease in which there is a diagnostic question remaining after physical examination, roentgen study and appropriate laboratory investigations have been carried out.

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19. Martin, G. E.: Value of Tracheobronchoscopy and Esophagoscopy in Diagnosis and Treatment, Edinburgh M. J. **48**:535 (Aug.) 1941.

20. Clerf, L. H.: Bronchoscopy as a Diagnostic Aid in Nontuberculous Pulmonary Disease, West. J. Surg. **49**:557 (Oct.) 1941.

Hemoptysis.—The determination of the cause and source of hemoptysis often presents great difficulty. Pitkin [21] reported the case of a patient aged 31 who first had a small pulmonary hemorrhage about one month before admission. Clinical and roentgen studies revealed no evidence of pulmonary disease, nor did the instillation of iodized oil exhibit bronchial obstruction or bronchiectasis. Several bronchoscopic examinations revealed nothing remarkable. Later at bronchoscopy blood was observed coming from the left lower lobe of the bronchus. There were repeated severe hemoptyses, some of which exceeded 1,000 cc. Pneumothorax was carried out to collapse the left lung, but in spite of this bleeding continued. It was finally decided on the basis of a surgical consultation that pneumonectomy would have to be resorted to, and the left lung was removed. Curiously, the histologic studies as well as the gross examination of the lung revealed remarkably little. There were observed recent and remote hemorrhages into the alveoli of the left lung with slight fusiform bronchiectasis.

Tracheobronchial Tuberculosis.—Unexplainable variations in the volume and consistency of the daily sputum of a patient with pulmonary tuberculosis arouse suspicions of a tracheobronchial obstruction with retention of the sputum, according to Becker and Holmes.[22] They found that the incidence of tracheobronchial obstruction revealed at autopsy in cases of pulmonary tuberculosis varied from none to 25 per cent in different parts of the world.

By bronchoscopic investigation of 25 tuberculous patients whose roentgenograms suggested the presence of collapse, Morlock [23] found definite tuberculous obstruction in 19. Apart from the discrete ulcer any of the forms of tracheobronchial tuberculosis were found to give rise to obstructive emphysema. In all of these cases the bronchial obstruction occurred in the larger bronchi. The author reasoned that the same process is active in the smallest bronchi and bronchioles and plays a part in determining the morbid anatomic changes of pulmonary tuberculosis and therefore the physical signs of the disease. Morlock also expressed the opinion that in the future in all diseases of the lung less concern should be paid to infiltration and consolidation and more to bronchial obstruction, whether partial or complete. Bronchial obstruction may cause difficulty in the diagnosis of tuberculosis by rendering the sputum free of tubercle bacilli although there are definite physical and

21. Pitkin, C. E.: Repeated Severe Hemoptysis Necessitating Pneumonectomy, Ann. Otol., Rhin. & Laryng. **50**:914 (Sept.) 1941.

22. Becker, P. H., and Holmes, G. W.: Tracheobronchial Obstruction and Its Relation to Pulmonary Tuberculosis, J. Indiana M. A. **34**:447 (Sept.) 1941.

23. Morlock, H. V.: Bronchial Obstruction in Pulmonary Tuberculosis, Tubercle **22**:207 (Sept.) 1941.

roentgenographic signs of pulmonary tuberculosis. In the absence of sputum containing the organisms the diagnosis remains in doubt until bronchoscopy reveals the presence of the obstruction.

Two patients with unexpandable lungs and tuberculous bronchial stenosis have been described by Farber.[24] Reexpansion of the lungs could not be obtained by bronchial dilatation, and this condition was considered to be due to extensive intrapulmonary fibrosis. A significant part of this parenchymal scarring followed nonspecific pneumonitis, which in turn was caused by the nonocclusive tuberculous bronchial stenosis.

Three personally observed cases of tuberculous obstructive emphysema and 16 cases published by other observers were reviewed by Barsby.[25] The pulmonary emphysema was due to bronchial narrowing which was caused by the enlargement of the lymph glands that is characteristic of a primary tuberculous infection. The author classified three stages of the disease depending on the degree of mediastinal displacement. In all cases the disease occurred in infants and children, and it was observed most often in children between the ages of 6 months and 1 year. Bronchoscopy showed either an extrabronchial mass protruding into the bronchial lumen or tuberculous granulation tissue to be the cause of the obstruction. These patients were benefited by bronchoscopic treatment, and it was necessary in some of them to reestablish the airway by removal of a portion of the obstructing lesion.

Although tuberculosis of the trachea and bronchi has been recognized by pathologists for many years, the subject has but recently been brought to the attention of clinicians. This is in great part due to the contributions made by bronchoscopy in this field. Myerson[26] reported his observations in a series of 572 consecutive bronchoscopic examinations of tuberculous patients. A specific lesion was found in the trachea or bronchus of 152 of these. This gave an incidence of tuberculosis of 4 per cent.

Obviously the same bronchial lesions are not seen by the pathologist and the bronchoscopist. While the pathologist sees only the last changes which have occurred, the bronchoscopist can observe the entire life picture of a tuberculous lesion. He, however, is handicapped in that he sees only the proximal portion of the lesion and is unable to give an opinion concerning the more distal portions of the lung in which the tuberculous disease is present.

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24. Farber, J. E.: Bronchial Stenosis and Unexpandable Lungs: Report of Two Cases, Am. Rev. Tuberc. **43**:779 (June) 1941.

25. Barsby, B.: Tuberculous Obstructive Emphysema in Children, Lancet **1**:627 (May 17) 1941.

26. Myerson, M. C.: Tuberculosis of the Trachea and Bronchus, J. A. M. A. **116**:1611 (April 12) 1941.

Myerson expressed the opinion that all bronchial tuberculosis with the exception of that due to penetration into the airway of a disintegrating lymph node originates from a cavity in the lungs. This was observed in a large proportion of autopsies performed in cases of fatal tuberculosis. A bronchial lesion commonly begins as a submucous infiltration which causes a slight elevation of the mucosa. This is an early lesion and is not frequently encountered by the bronchoscopist. It may remain in the submucosa and undergo resolution; it may become progressive and extend to other parts of the bronchial wall, or it may extend only to the surface. When this breaks through an intact mucosa ulceration results, with the formulation of a granuloma which is described as an ulcerogranuloma. This type of lesion, which is relatively common, was seen in 101 cases. It represents the most active phase of the disease, and on filling the adjacent branch bronchi atelectasis of the lung distal to it occurs. In the healing of the ulcerogranulomatous lesion the extent of fibrosis is dependent on the degree of involvement of the bronchial wall itself and may consist of little more than a superficial fibrous change or there may be marked cicatricial stenosis.

The common indications for bronchoscopy in the tuberculous patient are: a routine procedure before thoracoplasty; the presence of tubercle bacilli in the sputum associated with a pulmonary lesion, which should have been controlled by the therapeutic measures carried out; the spread of the disease to a healthy lung, which was not expected on the basis of the existing pulmonary condition; wheezing or noisy breathing; unexplained atelectasis, and the presence of tubercle bacilli in the sputum despite apparently normal roentgenographic observations.

Kernan and Cracovaner [27] reported their results in the treatment of 56 patients with tuberculosis of the trachea and bronchi. In the great majority the lesion consisted of ulceration or granulation tissue, while the remaining 6 had fibrous strictures or swollen mucous membranes. The symptoms which called for bronchoscopy were, in order of frequency, wheezing, persistent tubercle bacilli in the sputum, hemorrhage, severe dyspnea and persistent cavity. The methods of treatment employed were irradiation with a quartz mercury vapor lamp, cauterization with 10 per cent silver nitrate and, in only 3 cases, diathermy coagulation. The authors considered 35 patients cured so far as the endobronchial lesion was concerned. In 9 cases the treatment was a complete failure. In 3 cases there was improvement, but it was not adequate and treatment was suspended. Eight patients showed improvement at the time of their last treatment, but for some reason they did not return for further

27. Kernan, J. D., and Cracovaner, A. J.: Results of Treatment of Tuberculosis in Trachea and Bronchi, New York State J. Med. **41**:482 (March 1) 1941.

observation. In this series 4 had recurrence of the ulceration which was healed by further treatment, but 3 others had recurrence which failed to heal when further treatment was instituted. Two of them had a spread of the infection following bronchoscopy. The condition in one of these eventually became entirely healed, while the other patient died of miliary tuberculosis. In the latter case autopsy showed the bronchus to be healed. One patient died after bronchoscopy when a profuse hemorrhage developed on cauterization of an ulcerative lesion.

Pulmonary Abscess.—Anatomic pulmonary units were termed bronchopulmonary segments by Glass.[28] They are useful for clinical localization of pulmonary abscess, bronchiectasis and bronchopneumonia. A series of 100 cases of acute abscesses of the lung was analyzed according to bronchopulmonary segments involved and clinical location. The subdivisions of the lobar bronchi are constant in number and position, and the mass of pulmonary parenchyma aerated by each subdivision has a constant shape, size and position in the thoracic cage. The author stated that larger putrid pulmonary abscesses always lie in the periphery of the lobe, their most superficial portion being within ¼ or ½ inch (0.6 to 1.3 cm.) of the surface. The superficial situation is due to the onset of the disease in the finer bronchioles, but the abscess always lies in direct line with certain large bronchi. In 40 per cent of the cases the abscess was located in the apical segment of the lower lobe.

A series of 25 consecutive cases of abscess of the lung treated by surgical drainage was presented by Betts.[29] Inhalation of material from the upper respiratory passages appeared to be the causative factor in the majority of cases.

Moersch and Olsen[30] treated 193 patients with putrid pulmonary abscesses by bronchoscopic drainage. Satisfactory results were listed in 65.5 per cent of the cases. In 5 of the 9 patients who died empyema was associated with the abscess and probably bronchoscopy should not have been performed. Pneumothorax and pulmonary edema were the cause of death of the other 4 patients. Only 1 patient classified as having been treated successfully suffered a recurrence, and the second abscess was located in the opposite lung. Tonsillectomy with the patient under general anesthesia was the most frequent cause for pulmonary abscess, but no correlation could be found between the cause of the abscess and its prognosis. The earlier in the course of development of

28. Glass, A.: Abscess of Lung: Bronchopulmonary Segment, Basis for Clinical Localization, Surgery **10**:465 (Sept.) 1941.

29. Betts, R. H.: Principles in Management of Pulmonary Abscess, Am. J. Surg. **54**:82 (Oct.) 1941.

30. Moersch, H. J., and Olsen, A. M.: Role of Bronchoscopy in Treatment of Pulmonary Abscess, Surgery **9**:905 (June) 1941.

the acute abscess that bronchoscopy was carried out the better the prognosis. The authors' procedure has been absolute rest and postural drainage for the first two weeks. If at the end of this time resolution has not begun bronchoscopic aspiration is proceeded with. If the region cannot be drained adequately or if some satisfactory improvement does not take place after two or three bronchoscopic aspirations, surgical intervention is advised.

To treat pulmonary abscess successfully it is necessary to recognize various predisposing, etiologic, bacteriologic and pathologic factors which must be considered in the production of pulmonary abscess. Spector [31] reported on 2 patients with pulmonary abscesses, one bilateral and multilobar and the other unilobar, both of whom were treated with sulfanilamide with complete recovery. The predominating etiologic organism was a hemolytic streptococcus. In both cases large doses of sulfanilamide were used, leading to a high concentration in the blood. It is of interest to note that there was a marked difference in the mechanism in the production of the abscesses although the organisms were identical. In one case the abscess probably was of hematogenous origin, while in the other it occurred by the aspiratory route.

Two cases of amebic abscess of the liver with pleuropulmonary symptoms and complications were reported by Sherman and Weinberger.[32] In one case the pulmonary abscess with amebiasis was cured by specific treatment with emetine hydrochloride and acetarsone. A large solitary amebic abscess of the liver without evident pulmonary disease but characterized by cough, sputum and dyspnea was present in the remaining case.

Bronchiectasis.—An analysis of 116 cases of bronchiectasis with regard to the relation between the causes, symptoms, signs and type of dilatation observed on roentgenographic study was made by Chapman and Hoskins.[33] There were 60 cases of cylindric, 26 of saccular, 24 of varicose, 4 of fusiform and 2 of bronchiolectatic types of bronchiectasis. Hemoptysis was more common with the saccular type. The incidence of pneumonia in saccular bronchiectasis was greater than in the cylindric type, with about 40 per cent of the patients asserting that they had never had the disease. Nasal disease was a complaint of 35 per cent, and its incidence was about the same with the three main types of bronchiectasis.

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31. Spector, H. I.: The Treatment of Multiple Abscesses of the Lung with Sulfanilamide: A Report of Two Cured Cases, Dis. of Chest **8**:7 (Jan.) 1941.

32. Sherman, G. A., and Weinberger, H.: Amebiasis with Pleuropulmonary Complications: Report of Two Cases, J. Michigan M. Soc. **40**:289 (April) 1941.

33. Chapman, J., and Hoskins, H.: Bronchiectasis: Morphologic Varieties of Bronchiectasis in Relation to History, Symptoms and Signs, Am. Rev. Tuberc. **43**:512 (April) 1941.

Physical signs in the cylindric type were absent more often than in the other types, but the only patients known to have asthma were in this group. The authors stated the belief that the distinction between cylindric and saccular bronchiectasis is based on sound grounds, but this is not the case with the other morphologic types.

Chapman and McCullough [34] classified chronic bronchiectasis into four main groups according to etiology, anatomy, function and prognosis. The etiologic factors were subdivided into congenital, primary and acquired. The anatomic factors consisted of location, morphology, degree of dilation and associated pathologic changes. According to the functional factor the disease was considered symptomless, dry, mildly or severely productive and septic. The prognosis concerned progression, nonprogression and danger to health and life.

In the experience of Schenck [35] patients with chronic disease of the lower part of the respiratory tract and purulent sputum in large amounts showed an overwhelming incidence of chronic sinus infection. Of those studied by him 90 per cent showed roentgenographic indications and 80 per cent clinical evidences of active disease of the sinuses. The average number of sinuses involved is very high, and in most instances there is pansinusitis. Experimental evidence supports the belief that sinus infection is primary and bronchial infection secondary, and, further, it indicates that infection within the sinuses may extend to the lower part of the respiratory tract by inhalation, by way of the lymphatics or by an indirect route, in which the submucosal lymph structures first become involved and later infected secretions from the mucosal surfaces reach the lower respiratory tract by inhalation.

From a practical standpoint it is obvious that the treatment of chronic suppurative disease of the lower respiratory tract is doomed to failure if it does not include thorough treatment of the infection of the nasal sinuses. Arrest of the pulmonary lesion will not always occur after the diseased sinuses are cured, and once the bronchial disease is well established eradication of the sinus infection will not arrest the bronchial disease.

Bronchoscopy and bronchography have changed completely the conception of bronchiectasis, and by their use it is now possible to recognize types of disease in which characteristic symptoms may be absent. Leopold [36] expressed the opinion that these diagnostic methods have

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34. Chapman, J., and McCullough, D.: Classification of Chronic Bronchiectasis, Am. Rev. Tuberc. **43**:521 (April) 1941.

35. Schenck, H. P.: Etiology of Bronchiectasis, Arch. Otolaryng. **34**:958 (Nov.) 1941.

36. Leopold, S. S.: Bronchiectasis: Medical Aspects, Arch. Otolaryng. **34**:966 (Nov.) 1941.

made it apparent that in frequency bronchiectasis stands second only to tuberculosis among chronic diseases of the lung. He discussed the role of certain infections as etiologic agents and the influence of bronchial obstruction in the production of bronchiectasis. He stated the belief that the medical treatment of bronchiectasis is effective only before the pathologic changes of the bronchi become irreversible; therefore early diagnosis is essential for effective conservative therapy.

Bronchiectasis commonly is a sequel or complication of the pulmonary phase of the acute infectious diseases of childhood. While the part played by obstruction of the bronchus in the development of bronchiectasis was not fully understood until it was demonstrated by bronchoscopic observations, it is now a well known fact that bronchial obstruction is an important etiologic factor. Tucker [37] pointed out that bronchial obstruction which is relieved before infection and inflammatory changes have occurred in the bronchial wall usually will not be complicated by bronchiectasis, whereas if the obstruction is of long standing and there is suppuration beyond, the likelihood of dilatation of bronchi is greatly increased. It may be necessary under such circumstances to repeat bronchoscopic aspiration of secretions so that ventilation and drainage of the involved bronchi are improved. Infection due to obstructing secretion, the products of bronchial and pulmonary infection, drowned lung and unresolved pneumonia may be effectively removed with the aid of the bronchoscope.

In bronchiectasis occurring as a complication or sequela of infection of the upper respiratory tract the treatment of an abnormal condition of the sinus alone will not cure the diseased lung. Investigation and treatment of the bronchiectasis by bronchoscopy must be carried out coincidentally with the treatment of the sinus disease if this is indicated. The function of the bronchoscopist is to render every possible aid to the otolaryngologist, internist, pediatrician, roentgenologist and thoracic surgeon in the diagnosis, prevention and cure of bronchiectasis.

Since bronchiectasis is a disease which carries a high mortality rate over a period of years if untreated or inadequately treated, Johnson [38] agreed that radical surgical procedures offer an excellent chance of complete cure with a minimal mortality in patients with unilateral bronchiectasis. Although the risk in patients with bilateral bronchiectasis is increased and the chances of a complete cure are decreased, nevertheless the condition of many of these patients may be greatly improved, and a few may be completely cured.

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37. Tucker, G.: Bronchoscopic Aspects of Bronchiectasis, Arch. Otolaryng. **34**:999 (Nov.) 1941.

38. Johnson, J.: Surgical Treatment of Bronchiectasis, Arch. Otolaryng. **34**: 991 (Nov.) 1941.

In a study of a series of patients with bronchiectasis, Riggins [39] agreed that congenital bronchiectasis is a very rare condition and probably is confused with the acquired form of saccular bronchiectasis. Bronchiectasis commonly has its onset in childhood or early adult life. On the other hand, chronic disease of the nasal sinuses usually has its onset relatively later in life. He stated the belief that chronic disease of the nasal sinuses is more often secondary to bronchiectasis than bronchiectasis is secondary to diseases of the sinuses. In a group of 100 cases studied from the standpoint of disease of the nasal sinuses it was found that 30 per cent of the patients had this condition. Of these, approximately two thirds had symptoms, signs or both of bronchiectasis for several months to several years before evidences of sinus disease developed.

While there is no satisfactory medical treatment for the cure of bronchiectasis, certain measures may aid in securing symptomatic improvement. While these measures give temporary relief they do not exert any permanent aid and a continuation of symptoms, notably cough and expectoration, are the rule. Chronic invalidism of a physical and psychologic nature, often lasting throughout an entire lifetime, with the ultimate prospect of pneumonia, pulmonary abscess, pulmonary hemorrhage, cardiac failure or some related complication, is a not uncommon picture in these cases and should always be taken into account by the physician as well as the surgeon when the question of surgical treatment of bronchiectasis is considered. Because of the very unfavorable outlook with all forms of medical treatment most patients with bronchiectasis should be considered as candidates for surgical treatment, although a considerable number will be found inoperable. In suitable cases lobectomy is a safe and curative procedure for bronchiectasis.

Regarding the importance of roentgen study in cases of bronchiectasis Pendergrass [40] discussed pertinent effects of the roentgenologic diagnosis and reviewed those phases of pulmonary dynamics and the roentgenographic appearances of structures that are modified by the diseased process. In considering bronchography he emphasized that patients suspected of having bronchiectasis should be submitted early to this procedure with the hope that a comprehensive diagnosis can be made before the final picture of bronchiectasis is initiated, before there is destruction of the muscle, cartilage and elastic fibers of the bronchial wall and when the disease is still reversible and therefore amenable to conservative treatment.

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39. Riggins, H. M.: Bronchiectasis: Morbidity and Mortality of Medically Treated Patients, Am. J. Surg. **54**:50 (Oct.) 1941.

40. Pendergrass, E. P.: Roentgenologic Considerations of Bronchiectasis, Arch. Otolaryng. **34**:974 (Nov.) 1941.

Although numerous contributions dealing with the treatment of bronchiectasis have appeared in the literature, therapy is far from satisfactory except in the small group of patients that can be treated by surgical means. Carpenter, Kerr and McMurray [41] reviewed the literature on roentgen therapy of bronchiectasis. They reported their results of treatment in a series of 38 cases and concluded that roentgen treatment of patients with chronic bronchiectasis has resulted in some symptomatic improvement in about 50 per cent of those in whom the disease was mild or moderately advanced and in a small percentage of those in whom the disease was far advanced.

Roentgen therapy may be a valuable adjunct in the treatment of patients who have had little improvement from treatment of the foci of infection by bronchoscopic drainage and from other forms of therapy and who are not amenable to surgical treatment. It also may be of value in the treatment of elderly patients with cardiac disease, patients for whom bronchoscopic treatment is contraindicated because of age or other conditions, persons who cannot follow a prolonged regimen of treatment and possibly young children.

Postoperative Pulmonary Atelectasis.—Although pulmonary emboli probably are responsible for some complications following operations, retained secretions in the bronchi can be blamed for most of the instances of pneumonia and atelectasis which occur during the postoperative periods. According to Haight and Ransom,[42] the presence of bronchial secretion after operation is due to preexisting inflammation of the tracheobronchial tree, entrance of secretions from the upper air and food passages into the bronchial tree during or following an operation or formation of secretions postoperatively. In their experience these complications developed more commonly in male patients, and they explained this on the basis that males, being abdominal breathers, are prone to have excessive pharyngeal or bronchial secretions.

As a prophylactive measure it is important to observe whether there is present an acute or chronic inflammatory disease of the upper air or food passages or tracheobronchial tree prior to operation or whether there is an excess of pharyngeal or bronchial secretion present. In order to prevent aspiration, Haight and Ransom recommended that preoperative administration of large doses of sedatives and atropine be abolished. The operating table should, if possible, be inclined to a 10 or 15 degree Trendelenburg position. The question of anesthesia is probably of little consequence, for as many pulmonary infections occur after inhalation as after spinal or other forms of anesthesia.

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41. Carpenter, R. C.; Kerr, H. D., and McMurray, J. S.: Roentgen Therapy for Bronchiectasis, Arch. Otolaryng. **34**:913 (Nov.) 1941.

42. Haight, C., and Ransom, H. K.: The Prevention and Treatment of Postoperative Atelectasis and Bronchopneumonia, Ann. Surg. **114**:243 (Aug.) 1941.

In the postoperative care it is important to have the patient return to consciousness promptly after operation. The foot of the bed should be elevated, and the patient should be turned from side to side at frequent intervals. Frequent examination of the chest also should be made in order to ascertain if there are evidences of hypoventilation. If there is accumulation of secretions the authors recommend drainage of the bronchi by suction, either through a bronchoscope or through a catheter if other measures are not successful. While bronchoscopy permits of shrinking the mucosa with topical applications, thereby enlarging the bronchial lumen, the catheter method probably is more readily applied. They employed a no. 16 catheter which has two openings. This is inserted through the nose into the trachea during deep inspiration, and with the aid of negative pressure secretions are aspirated from one and then from the other main bronchus. This may be repeated as often as is necessary, but as the condition of the patient improves voluntary coughing usually takes care of any secretions which remain or reform. If this relatively simple measure is carried out at the earliest indication of retention of secretions, the incidence of pneumonia and atelectasis will be reduced.

Apfelbach and Carter [43] reviewed the mechanism of postoperative atelectasis and have not entirely accepted bronchial obstruction as the primary etiologic factor. They stated the belief that there may be a reflex mechanism from the vegetative nervous system producing a vago-sympathetic block which is responsible for the pulmonary collapse.

Fisher [44] reported 9 cases of acute massive pulmonary collapse which resisted conservative treatment but responded promptly to bronchoscopic aspiration. Bronchoscopy is advised if mucus cannot be expelled from the lung by stimulation of the cough reflex with inhalations of carbon dioxide and oxygen or by turning the patient from side to side and manipulating the chest wall.

In an analysis of 30 cases of postoperative pulmonary atelectasis in which bronchoscopic treatment was given, Lell [45] agreed that palliative measures, such as hyperventilation, postural drainage and stimulation of the cough reflex, should first be tried and if these are unsuccessful then bronchoscopic drainage should be carried out. The employment of morphine and other sedatives that inhibit the cough reflex should be avoided. It was interesting to note that in the 30 cases local anesthesia was employed in 9, ether anesthesia in 3 and gas-ether anesthesia in

43. Apfelbach, G. L., and Carter, J. B.: Recurrent Postoperative Atelectasis, Radiology **37**:598 (Nov.) 1941.

44. Fisher, G. E.: Recognition and Bronchoscopic Treatment of Pulmonary Atelectasis, J. M. A. Alabama **10**:340 (April) 1941.

45. Lell, W. A.: Bronchoscopy in the Treatment of Postoperative Pulmonary Atelectasis, Pennsylvania M. J. **44**:1551 (Sept.) 1941.

the remaining cases, in 4 of which this was supplemented by spinal anesthesia. In 22 cases only one bronchoscopic aspiration was necessary, while in 4 it was carried out twice and in 2 it was necessary to do bronchoscopy three times. One patient had five aspirations and 1 had eight bronchoscopies; these 2 eventually died.

Adenoma of the Bronchus.—While polypoid bronchial tumors may include carcinoma, adenoma and purely local tumors, such as fibroma, lipoma and myoma, the bronchial adenomas do not metastasize distantly and are locally invasive. They therefore can be cured by surgical removal. In a discussion of this subject, Goldman and Stephens [46] reviewed the extensive literature and suggested a more comprehensive classification. They reported on a group of 18 patients with bronchial adenoma, of which 11 were females and 9 males.

The symptoms and clinical course of bronchial adenoma differ from those of carcinoma. There are three peculiarities to the life history of this tumor: adenoma commonly is located in a major bronchus, its growth is relatively slow and it is extremely vascular. One of the outstanding symptoms therefore is pulmonary hemorrhage. This usually is sudden in onset as well as in termination; the amount of bleeding often is profuse and is unprovoked by cough or exercise, and in women it frequently occurs during the menstrual period. The hemorrhage apparently arises from the tumor itself. In addition to hemorrhage one also observes the symptoms common to bronchial obstruction which is slow but progressive. These include wheezing respiration, a nonproductive irritative cough, transient pain in the chest and often dyspnea. Later, when the tumor becomes obstructive, atelectasis results, and then there occur the symptoms and signs commonly associated with complete bronchial obstruction. The bronchoscopic appearance of adenoma is usually sufficiently distinctive to differentiate it from primary bronchial carcinoma. There is no ulceration or infiltration of the bronchial mucosa. The growth itself is freely movable unless it is firmly impacted in the bronchus. Hemorrhage is of common occurrence and is observed immediately on even slight trauma to the growth. Since adenoma may appear as an endobronchial as well as an extrabronchial tumor the extent cannot always be determined on the basis of simple roentgen study or bronchoscopy alone. Tomography is very useful in the diagnosis of the extent of the growth.

With regard to treatment, endobronchial lesions usually can be taken care of satisfactorily by bronchoscopy alone. If there is an extrabronchial portion it may be necessary to resort to external surgical extirpation. In addition, if there is pulmonary suppuration distal to the obstruction surgical treatment is indicated.

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46. Goldman, A., and Stephens, H. B.: Polypoid Bronchial Tumors with Special Reference to Bronchial Adenomas, J. Thoracic Surg. **10**:327 (Feb.) 1941.

Although there is considerable confusion concerning the histologic classification of the so-called adenoma of the bronchus, early diagnosis is important. If the tumor is benign it ultimately will produce bronchial obstruction with suppuration distal to the growth unless the drainage and ventilation of the lung distal to it can be maintained. If it proves to be malignant prompt diagnosis is necessary if surgical extirpation is to be considered. Jackson and Konzelmann [47] discussed the diagnostic criteria of adenoma and pointed out that hemoptysis is probably the most common symptom, although wheezing, productive cough and dyspnea frequently are observed. The clinical and roentgenologic observations are commonly those of obstructive emphysema or atelectasis, although many patients present few symptoms or physical signs in the early stages. In the authors' opinion bronchoscopic treatment is effective in most cases of bronchial adenoma as well as in cases of other benign bronchial tumors, but often a number of treatments are necessary. In some cases it seems impossible to obtain a permanent cure, such as would make subsequent treatments unnecessary. They believe therefore that conservative measures should be given a trial in every case but that surgical treatment of a more radical character should be considered if conservative treatment is ineffective. They presented follow-up data on a series of cases previously reported and added 1 additional case.

Carcinoma of the Bronchus.—In reflecting on the attitude of the laity as well as of the average physician toward bronchogenic carcinoma, Gebauer [48] pointed out that this attitude will continue as long as it is believed that the absence of bronchoscopic evidence rules out cancer in from 75 to 85 per cent of cases and as long as roentgen examination is delayed until physical signs appear. He emphasized that cancer of the lung cannot be regarded pathologically or clinically as a single entity, that bronchoscopy is of definite value in the early stages and also that time is precious in the recognition of this condition, for in many there is no significant silent period and symptoms tend to occur early. He discussed the pathologicoclinical and the bronchoscopic differentiation of the tumors. In his series positive histologic evidence was obtained in 72 per cent of more than 100 bronchoscopies and a diagnosis of carcinoma was made in 87 per cent of the cases.

When one considers the frequency with which a diagnosis of cancer is made by bronchoscopy it is obviously important to consider the duration of symptoms. If bronchoscopy is performed early in the disease the frequency with which biopsy can be done will be much lower than if bronchoscopy is performed in the advanced stages. In Gebauer's

47. Jackson, C. L., and Konzelmann, F. W.: So-Called Adenoma of the Bronchus, Ann. Otol., Rhin. & Laryng. **50**:1264 (Dec.) 1941.

48. Gebauer, P. W.: The Differentiation of Bronchogenic Carcinomas, J. Thoracic Surg. **10**:373 (April) 1941.

series much of the clinical material was practically autopsy material, for the average duration of life from admission to death was only one month. The value of bronchoscopy in a study of tumor incidence can be based only on those cases in which the tumor appears in bronchi within range of the bronchoscope. In this series 35 per cent of the tumors arose from a main bronchus, 26 per cent in an upper lobe bronchus, 26 per cent in a lower lobe bronchus and 13 per cent in the middle lobe bronchus or in small branch bronchi. Few of the tumors in the upper lobe, none of the tumors of the branch bronchi and not all of the tumors in the lower lobe would be visible bronchoscopically if observed early in their course. It is probable, therefore, that bronchoscopy would have shown no abnormalities in about 40 per cent of cases if these had been examined early in the disease.

Diamond [49] expressed the opinion that the diagnosis of primary carcinoma of the lung cannot be made with assurance at the initial examination but only after a painstaking and systematic study. Data gathered by careful history taking, physical examination and one or more roentgenograms determined the nature of the subsequent procedures. Even before physical and roentgenographic signs of pulmonary disease have appeared and in the presence of unexplained symptoms bronchoscopy should be performed. The author advocated the instillation of iodized oil in the bronchial tree before diagnostic bronchoscopy, although he stated that this is not essential for diagnosis. In cases in which a tumor is inaccessible bronchoscopically and a specimen for biopsy cannot be obtained the creation of an artificial pneumothorax with thoracoscopic biopsy is indicated. The final diagnosis of carcinoma of the lung can be made only histologically, and every effort should be made to secure a section of tissue for examination.

Ormerod [50] reported 100 cases of histologically proved carcinoma of the bronchus treated by the application of radon. Life expectation was advanced by radon treatment from three and five-tenths to five and six-tenths months for the 100 patients, though as several of these patients are still alive the average is being gradually increased. The author pointed out that in many cases the disease was far advanced and the treatment considered palliative only.

In treating such a disease as cancer of the bronchus the survival rate of patients is perhaps not so immediately important as the very satisfactory results in certain cases. In this series 21 have survived more than a year, 12 for one to two years, 5 for two to three years, 1 for over three years, 1 for over four years, 1 for five years and 1 for eight

49. Diamond, S.: Clinical Diagnosis of Primary Carcinoma of Lung, Am. Rev. Tuberc. **43**:713 (June) 1941.

50. Ormerod, F. C.: Some Notes on Treatment of Carcinoma of Bronchus, J. Laryng. & Otol. **56**:1 (Jan.) 1941.

years. Of these 21 patients all except 3 were under 60 years of age; 2 of the 3 were aged 60 and the other 62. There was only 1 under 40 years of age. Of the patients still alive 1 was treated over eight years ago and was then 36 years old; 1 who has been well for over five years was 49 when first seen. Three who have been well for over two years were 55, 54 and 49 respectively. From the figures it is evident that for the more elderly patients who do not respond well to treatment and for those over the age of 60 only alleviation of symptoms and not any real degree of cure can be expected. With this exception there does not seem to be any factor which can indicate in which cases cancer of the lung is likely to respond to radon treatment, and selection is therefore made impossible.

It would be inappropriate to apply radon intrabronchially in cases in which there was a reasonable prospect of successful surgical removal. It is not of value to apply such treatment when there is very advanced disease, as indicated by lymph node involvement or metastasis. Opening up of the bronchus, evacuation of purulent secretions, reduction of toxic absorption and improved aeration of the lung are of benefit. In addition to this, 1 patient in 5 will survive more than a year and 1 in 10 will survive more than two years, while a certain number may be regarded as cured. According to the technic used by Ormerod, the first application of radon is made a week or more after the diagnostic bronchoscopy. The dose of radon used is usually ten silver seeds of 3 millicuries each. These are inserted as nearly as possible into the attached portion of the tumor or at the point at which the tumor has penetrated the wall of the bronchus to reach the lumen. Besides being introduced into the attachment of the growth, seeds also may be inserted into the wall of the bronchus and if necessary through it. A roentgenogram is taken shortly after implantation to show the position of the seeds. Very little reaction usually follows the treatment, but the patient remains in the hospital for two to three days. Two months after the first application of radon the patient returns for a second treatment. Subsequent applications are determined by the condition of the patient and the course of the disease. A satisfactory result consists in disappearance of the tumor formation from the bronchial lumen with reopening of the air passages and a reaeration of the corresponding pulmonary field. In some cases this is obtained, but in many there remains some fibrous stenosis of the bronchus at the site of the growth, preventing reexpansion of the lobe or portion of the lobe. The failure of the lung to reexpand does not seem to diminish the completeness of the recovery.

Bronchoscopy should be considered in every case of unexplained pulmonary symptoms. Betts [51] presented a series of 62 verified pul-

51. Betts, R. H.: Carcinoma of the Lung: Bronchoscopic Aspects, New England J. Med. **225**:519 (Oct. 2) 1941.

monary tumors; in this group the diagnosis was established by bronchoscopic biopsy in 46 cases, or 74 per cent. A roentgenologic diagnosis of pulmonary cancer is not sufficient, since a verified diagnosis cannot be made by this method. The extent or operability of a lesion may be misleading by roentgenography. Bronchoscopy is the most accurate way of determining these factors. A small submucous bronchial neoplasm may not produce pulmonary signs or symptoms, yet the lesion may be visible bronchoscopically.

From the bronchoscopic viewpoint, pulmonary carcinomas may be conveniently divided into two groups, central and peripheral. The central, or stem bronchus, group includes those lesions arising in the main bronchus, in the lobar bronchi or in the proximal portion of one of the lobar bronchial subdivisions. If the tumor itself can be seen endoscopically, it necessarily falls in the central classification. Lesions in this central group comprise approximately 75 per cent of all primary neoplasms. Peripheral tumors cannot be directly visualized by bronchoscopy, although frequently a deformity of the bronchus may be noted. Such an observation is strong presumptive evidence of neoplasm, although tissue may not be obtained for histologic examination. Bronchoscopic evidence of inoperability may be grouped under two headings: direct local intrabronchial extension to an unresectable area of the tracheobronchial tree and extensive involvement of the paratracheal lymph nodes.

Therapeutically bronchoscopic dilatation or even removal of a portion of the tumor mass provides better drainage and may give relief from the septic process. For certain lesions not amenable to surgical therapy, radium or radon may be used in an intrabronchial applicator introduced through the bronchoscope.

On the basis of a statistical analysis as well as an analysis of their own material, Menne and Anderson [52] expressed the opinion that there is both a relative and an absolute increase in the incidence of bronchogenic carcinoma. In a study of a series of 84 patients, it was noted that approximately half the patients with highly nodular carcinoma presented symptoms referable primarily to the lungs, while an equal number had symptoms attributable to metastasis. In the 38 per cent of the patients with the hilar nodular type of growth there were clinical evidences of atelectasis. Cough with expectoration was the most frequent symptom noted, occurring in two thirds of the cases. Dyspnea was the second most common symptom. Emphasis was placed on the etiologic significance of inflammatory processes augmented by volatile irritants, such as tobacco smoke and gaseous products of the industries.

52. Menne, F. R., and Anderson, M. W.: Bronchogenic Carcinoma: Incidence in the Pacific Northwest with a Commentary on Eighty-Four Cases, J. A. M. A. **117**:2215 (Dec. 27) 1941.

In discussing carcinoma of the lung Ochsner and DeBakey [53] stated that recently, because of early diagnoses as well as improvement in surgical management, the prognosis of pulmonary carcinoma has become relatively favorable. In their opinion the only factor that can explain the steady increase in incidence of pulmonary cancer is the increase in smoking, particularly cigaret smoking, which is universally associated with inhalation. Early diagnosis is essential to effective treatment and can be secured only by prompt investigation by bronschoscopy and roentgen study of any respiratory manifestation in a patient past 40 years of age. There are no characteristic symptoms of bronchial carcinoma, and the most important factor in the diagnosis is the consideration of its possible presence. The incidence of correct diagnoses made by bronchoscopic visualization of the lesion will probably be higher when made by one experienced in the bronchoscopic diagnosis of this tumor than by routine microscopic examination of biopsy specimens.

Bronchoscopy is also of value to the surgeon in determining the extent of the lesion. In those instances in which there is compression of the trachea or in which there is marked widening of the carina the lesion is likely to be inoperable.

Ochsner and DeBakey [54] reviewed the incidence, diagnosis and treatment of primary carcinoma of the lung. They found that primary carcinoma of the lung now ranks second as the primary site of cancer. It occurs predominantly in the male sex, is most frequently encountered in the fifth and sixth decades and should be suspected in every patient past 40 years of age who complains of cough that cannot be explained and that does not readily respond to therapy. Roentgenography and bronchoscopy are valuable diagnostic procedures and will provide an accurate diagnosis in over 70 per cent of the cases. Bronchography and aspiration biopsy are other valuable diagnostic aids. The only curative treatment is surgical extirpation of the involved lung.

A series of 38 cases of primary bronchogenic carcinoma of the lung was reported by Olin and Elliott.[55] They concluded that this disease still remains a problem as to early diagnosis and treatment, and too many cases are first observed when the tumor is well advanced. Emphasis was laid on early bronchoscopic examination and roentgen studies, biopsy of a lymph node and early examination for cancer cells in pleural fluid, sputum and material from puncture of the lung.

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53. Ochsner, A., and DeBakey, M.: Carcinoma of the Lung, Arch. Surg. **42**: 209 (Feb.) 1941.

54. Ochsner, A., and DeBakey, M.: Primary Carcinoma of Lung, New Orleans M. & S. J. **93**:387 (Feb.) 1941.

55. Olin, H. A., and Elliott, C. A.: Primary Bronchogenic Carcinoma of Lung: Clinical and Roentgen Study of Thirty-Eight Cases Over Period of Twelve Years, Am. J. Surg. **54**:614 (Dec.) 1941.

When considering the diagnosis of bronchogenic carcinoma one commonly thinks in terms of bronchial obstruction, which usually is an early sign. The physical, roentgenologic and bronchoscopic observations commonly reported are dependent on these findings. In discussing this subject Holinger and Rigby [56] presented 3 cases of malignant bronchogenic tumors which gradually infiltrated both lungs to produce death without evidence of bronchial obstruction or pulmonary suppuration.

In a study of the symptoms it was found that cough, the only symptom which usually is found in early bronchogenic carcinoma, was present in each case. A study of the roentgenograms also failed to reveal any persistent pathologic changes on which a diagnosis of carcinoma could be made. In 1 case the roentgenographic appearances were so characteristic of fungous infection that this diagnosis was made without question. The isolation of fungi from the bronchoscopically removed secretions further tended to confuse the diagnosis. The bronchoscopic observations were essentially negative so far as a diagnosis of carcinoma was concerned. Of great significance was the histologic examination of the lungs. The metastatic spread of the carcinoma throughout both lungs was lymphatic in each case.

Although irradiation therapy for primary carcinoma of the lung has not been effective as a curative agent, Tenzel [57] asserted that its usefulness in palliation and in prolonging life should not be overlooked. In an analysis of 192 treated and untreated primary carcinomas of the lung which were examined post mortem, Tenzel stated the belief that there was ample evidence to indicate that radiation therapy had prolonged life. He found that the survival periods of irradiated patients averaged five months longer than patients not so treated. Twice as many irradiated as nonirradiated patients survived more than one year. Symptomatic relief was obtained by 45 per cent of the patients treated.

While metastatic implants of carcinoma throughout the lung are common, single metastatic nodules invading and occluding a large bronchus are rare. Raine [58] reported on a woman who had been treated surgically for an obstructive annular carcinoma of the sigmoid. She remained well for practically ten months, and then a cough developed, which persisted and increased in severity. There was occasional hemoptysis. Roentgenographic study of the chest revealed an increase in density of the middle and lower lobes of the right lung. Bronchoscopic examination revealed that the carina was sharp and of normal

56. Holinger, P. H., and Rigby, R.: Bronchogenic Carcinoma Without Bronchial Obstruction, Ann. Otol., Rhin. & Laryng. **50**:1270 (Dec.) 1941.

57. Tenzel, W. V.: Radiation Therapy in Carcinoma of the Lung, J. A. M. A. **117**:1778 (Nov. 22) 1941.

58. Raine, F.: Metastatic Carcinoma of the Lung Invading and Obstructing a Bronchus, J. Thoracic Surg. **11**:216 (Dec.) 1941.

appearance and motion. Below the orifice of the right upper lobe bronchus a large papillary mass filled the bronchial lumen. Biopsy revealed an adenocarcinoma. This was similar to the lesion observed in the sigmoid. Since there was some question whether the pulmonary lesion was metastatic and that it might possibly be primary in the bronchus, as there were no other demonstrable areas of metastasis in either lung, a right pneumonectomy was done. The patient made an uneventful convalescence, but at the time the case was reported some gastric distress had developed and it was presumed that she had metastasis near the liver.

Giant Cell Tumor of the Trachea.—In their report of a case of giant cell tumor of the trachea, Cid and Bonilla [59] emphasized the importance of cough with little or no expectoration, hemoptysis and wheezing respiration as clinical manifestations of partial obstruction of a bronchus. The roentgenograms made early in the course of the disease exhibited evidences of atelectasis of the middle and lower lobes of the right lung with deviation of the trachea to the right. Subsequent roentgen studies revealed that the atelectasis had disappeared and the trachea was in normal position. At bronchoscopy there was found a tumor mass in the right main bronchus. A specimen was removed for biopsy. This was followed by a violent hemoptysis, and immediately the patient became very dyspneic. Later there was massive collapse of the right lung, and he died the following day. Although this case is the only one of giant cell tumor of the trachea that has been reported in available literature, it is of interest because the signs, symptoms and roentgenographic findings were characteristic of those observed in cases of partial bronchial obstruction.

Hodgkin's Disease.—A case of mediastinal tumor (Hodgkin's disease) with perforation into the trachea was presented by Hammond.[60] A youth aged 15 suffered from attacks of dyspnea, wheezy breathing, orthopnea, cough with thick yellowish sputum and generalized pains in the chest. Roentgenographic studies of the chest revealed mediastinal changes on the right side which suggested a glandular enlargement. Bronchoscopy was not considered advisable at that time because of the patient's poor condition and marked dyspnea. Irradiation was carried out, with marked improvement. About three months later he returned with a recurrence of the former obstructive symptoms. Irradiation was again instituted with marked improvement, and then the advisability of bronchoscopic examination was considered. At bronchoscopy the lower

59. Cid, J. M., and Bonilla, J. L., Jr.: Giant Cell Tumor of the Trachea, J. Thoracic Surg. **11**:210 (Dec.) 1941.

60. Hammond, A. E.: Perforation of the Trachea by a Mediastinal Tumor (Hodgkin's Disease), Ann. Otol., Rhin. & Laryng. **50**:929 (Sept.) 1941.

tracheal lumen was found partially obstructed by a tumor mass attached to the right tracheal wall just above the carina. A portion of the tumor was pedunculated and moved to and fro with the air current. A large portion of the mass was removed with forceps for microscopic examination. The microscopic diagnosis was Hodgkin's disease. A second bronchoscopy was done several days later to remove more of the tumor for study and also to enlarge the airway. There was decided improvement in breathing. Hammond emphasized the importance of bronchoscopy in the establishment of the diagnosis in obscure cases of obstruction to the airway and also its importance in outlining the therapeutic procedures to be followed.

Sarcoidosis.—A case of generalized sarcoidosis in which signs and symptoms of bronchial obstruction were the principal features was reported by Benedict and Castleman.[61] Bronchoscopy revealed extensive intrinsic involvement of the bronchial mucosa consisting of reddening and edema with bleblike formations throughout accompanied by marked narrowing of the main bronchi. The diagnosis was confirmed by histologic examination of the lesion and marked clinical improvement followed bronchoscopic bougienage.

Bronchography.—Jarvis[62] has modified the catheter method of instilling iodized oil into the trachea for roentgen examination. A hole is made in an ordinary catheter 1½ to 2½ inches (3.8 to 6.3 cm.) from the end; a silk thread is then run through the lumen and out through the new hole and sutured to the catheter tip. In this way, by pulling the thread the tip of the catheter is made to bend at any desired angle and the oil is injected either with a syringe or by gravity.

THE ESOPHAGUS

Physiologic Studies.—Faulkner, Rodenbaugh and O'Neill[63] discussed the influence of emotions on esophageal function. They compared esophagoscopic and roentgenologic observations and found that unpleasant thoughts or emotions produced a marked spasm while opposite emotions brought forth relaxation. These findings were noted both by direct inspection through the esophagoscope and in the esophagus filled with barium sulfate.

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61. Benedict, E. B., and Castleman, B.: Sarcoidosis with Bronchial Involvement, New England J. Med. **224**:186 (Jan. 30) 1941.

62. Jarvis, J. R.: Method for Instilling Iodized Oil in the Trachea, Ohio State M. J. **37**:746 (Aug.) 1941.

63. Faulkner, W. B., Jr.; Rodenbaugh, F. H., and O'Neill, J. R.: Influence of Emotions on Esophageal Function: Comparison of Esophagoscopic and Roentgenologic Findings, Radiology **37**:443 (Oct.) 1941.

Congenital Malformations.—Ten cases of congenital tracheoesophageal fistula were cited by Gengenbach and Dobos.[64] Seven of the infants had a congenital atresia of the esophagus; the other 3 had little if any esophageal obstruction and could take their feedings from a bottle. In all cases the fistula was between the lower esophageal segment and the trachea or bronchus. This condition should be suspected in the newborn when attacks of choking, coughing and cyanosis occur. These symptoms are aggravated on swallowing liquids, and any fluid taken is almost immediately regurgitated through the mouth and nose. If a small catheter is passed it soon encounters an obstruction in the esophagus, which can be demonstrated by roentgenograms after the instillation of an opaque medium. Even though surgical intervention has been unsuccessful, it offers the only hope for the ultimate survival of the child. If the infant does not die almost immediately or during the first few days of life, death in cases of atresia of the esophagus is usually due to an aspiration pneumonia.

Herniation of the stomach into the thorax by way of the hiatus esophageus is encountered with increasing frequency as efforts to diagnose or suspect it clinically are intensified by more relevant information regarding the condition. In a series of 47 patients with nontraumatic esophageal hiatal hernia reported by Polley,[65] congenital short esophagus with partial or complete thoracic stomach was present in 14. In all the cases the condition was considered as actually or essentially congenital. Age and obesity are believed to be contributing factors in the presence of a congenital potentiality for herniation through the hiatus. The clinical evidence should be substantiated by roentgen examination and by esophagoscopy if the roentgenologic evidence is inconclusive or if surgical intervention is contemplated. While esophagoscopy gives information concerning the interior of the esophagus, particularly the presence of ulceration and the degree of stenosis, Polley believed that roentgenologic examination, being simpler and less objectionable to the patient than esophagoscopy, is sufficient and that endoscopic examination could be restricted to persons presenting inconclusive evidence.

In a discussion of esophageal neuroses, Beatty and Palmer[66] described two types that have been observed, namely, difficulty with food and difficulty in swallowing medication. There commonly is no history of trauma of the larynx or esophagus. There also is no spasm

64. Gegenbach, F. P., and Dobos, E. I.: Congenital Tracheoesophageal Fistula, J. Pediat. **19**:644 (Nov.) 1941.

65. Polley, H. F.: Congenital Short Esophagus with Thoracic Stomach and Esophageal Hiatus Hernias: Report of Forty-Seven Cases, J. A. M. A. **116**:821 (March 1) 1941.

66. Beatty, H. G., and Palmer, D. M.: Esophageal Neuroses, Ann. Otol., Rhin. & Laryng. **50**:1245 (Dec.) 1941.

at either the upper or lower end of the esophagus, nor can there be demonstrated any evidence of obstruction either at the aortic or bronchial narrowings. While some patients have exhibited evidences of being nervous and neurotic, others have not presented any of these. Before proceeding with any plan of therapy it is important to pass an esophagoscope in order to rule out any definite organic obstruction. If one is certain that the case is one of neurosis, often the passage of a large esophagoscope with little or no anesthesia will aid in restoring the psychic balance. These neuroses are usually not permanent. They can best be treated by a combination of psychotherapy and of common sense training in the effective use of the deglutition apparatus.

Esophageal Stenosis.—Camiel and Loewe [67] directed attention to a syndrome of stenosis of the upper part of the esophagus in a report on 30 patients with esophageal complaints. The lesion was located at or near the uppermost portion of the esophagus. The syndrome is characterized by dysphagia, paralysis of one or both vocal cords, overflow of ingesta into the tracheobronchial tree, pulmonary complications resulting from aspiration and frequently clubbing of the fingers. Most of the lesions studied were neoplastic, but this syndrome may occur from any combination of factors resulting in similar anatomic or physiologic dysfunction. The incidence of pulmonary complications and recurrent nerve involvement dropped sharply when the lower two thirds of the esophagus was involved. All patients with lesions in the upper part of the esophagus died of aspiration pneumonia. Aspiration, with its attendant complications, may be expected with any obstructive and disruptive lesion at the upper end of the esophagus or with a lesion in the hypopharynx or piriform sinus.

Cicatricial Stenosis.—Vinson [68] listed 3 cases of cicatricial stricture of the esophagus following vomiting intubation. The strictures occurred from healing of inflammatory changes in the esophagus subsequent to abdominal operations in which intubations were performed. Cicatricial stenosis following vomiting or the passage of stomach tubes is usually located in the lower third of the esophagus, is annular in distribution and involves only a small portion of the wall. The stricture is single and produces dysphagia within a month or two of the time of onset of the inflammation. In Vinson's cases swallowing was unimpaired after satisfactory dilatation of the strictures.

The most common single etiologic factor in cicatricial atresia of the esophagus is the ingestion of caustics, although it may occur during the

67. Camiel, M. R., and Loewe, L.: A Syndrome of Upper Esophageal Stenosis, Ann. Int. Med. **15**:63 (July) 1941.

68. Vinson, P. P.: Cicatricial (Benign) Stricture of Esophagus Following Vomiting and Intubation: Report of Three Cases, West Virginia M. J. **37**:349 (Aug.) 1941.

course of systemic diseases. Hara and Rosenvold [69] reviewed the endoscopic methods of treatment that have been employed in cases of this condition and reported the successful treatment of a patient aged 24 who accidentally swallowed sodium hydroxide eight years previously. There developed an atresia of the lower part of the esophagus. The cicatricial barrier appeared to be at least ½ inch (1.3 cm.) in length. The stricture was penetrated by employing a cystoscope as a retrograde esophagoscope and a full lumen esophagoscope which was inserted above. With the aid of double plane fluoroscopy the tips of the instruments were placed directly opposite each other, and then with the aid of a urethral catheter containing a wire stylet which projected from its end against the cicatricial barrier a cutting current originating in a diathermic instrument was employed. This fulgurated through the cicatricial partition and the catheter was then drawn upward through the esophagoscope. Retrograde dilatation was instituted, and the patient ultimately made a complete recovery. They believed that the substrictural attack seems to offer a greater margin of safety, since the walls of the esophagus are thicker in the lower segment, the approach is more axial and the instruments are less likely to invade the mediastinum.

Syphilis of the Esophagus.—Four cases of esophageal obstruction due to a gummatous lesion of syphilis have been reported by Kampmeier and Jones.[70] In 1 case the obstruction was due to a gumma of the esophagus, while in the remaining cases a gumma of the diaphragm produced the obstruction. In 3 instances the nature of the lesion was recognized and antisyphilitic treatment together with active dilatation of the stenosis offered relief. In 1 case, in which the condition was erroneously diagnosed carcinoma, the patient died without appropriate treatment. The differentiation of a gummatous lesion of the esophagus or diaphragm from obstruction due to carcinoma or cardiospasm can be made only by direct examination (esophagoscopy and biopsy) and observations of the response to treatment.

Antisyphilitic treatment not only may fail to relieve but may actually increase the obstruction. Because of resultant fibrosis and contraction of the scar tissue esophageal dilatation will be needed.

Varix of the Esophagus.—In discussing the treatment of esophageal varices by the employment of sclerosing solutions, Moersch [71] expressed the opinion that it might be found necessary to repeat the injections if

69. Hara, H. J., and Rosenvold, L. K.: Cicatricial Atresia of the Esophagus, Arch. Otolaryng. **34**:574 (Sept.) 1941.

70. Kampmeier, R. H., and Jones, E.: Esophageal Obstruction Due to Gummas of Esophagus and Diaphragm, Am. J. M. Sc. **201**:539 (April) 1941.

71. Moersch, H. J.: The Treatment of Esophageal Varices by Injection of a Sclerosing Solution, J.Thoracic Surg. **10**:300 (Feb.) 1941.

further bleeding should take place. It is still uncertain whether the injection should precede or follow splenectomy and ligation of the coronary vein. The procedure, however, seems to be worthy of serious consideration, as it offers a means of controlling bleeding that has not responded to any other form of therapy and thus may be successful in prolonging the life of the patient.

Since his previous report on the employment of a sclerosing solution in the treatment of esophageal varices, Moersch [72] has added reports on 10 additional patients. In discussing the mechanism involved in the production of varices, Moersch pointed out that theoretically the purpose of injecting a sclerosing solution is to block the free passage of blood into the esophageal veins and by so doing to stimulate the portal circulation to increase its efficiency in conveying blood back through the liver in a normal manner.

The instrumentarium employed for injection into the veins and the technic employed are described. He emphasized the importance of being certain that the needle is in the lumen of the vein. The sclerosing solution, a 5 per cent solution of sodium morrhuate, is injected slowly into the vein. The amount to be injected depends on the patient's reaction. It is well to limit the amount to be injected at the first treatment to make certain that the patient is not sensitive to the solution. The amount injected varies but usually is 5 cc., although as much as 9 cc. has been injected at one treatment without ill effects.

In 2 cases severe hemorrhage occurred on withdrawal of the needle from the vein. In one it was necessary to pack the esophagus with a gauze pack to control the bleeding. In the second case the bleeding was promptly controlled by the topical application of fer de lance snake venom.

In 5 cases the varices were attributed to splenic anemia or Banti's disease and in 5 to portal thrombosis. In the second group of 5 cases the varices had occurred in patients on whom splenectomy had not been performed. While sufficient time has not elapsed to determine accurately the efficacy of the injection method of treatment, Moersch's experiences have been encouraging and the patients have stated that their general health has been improved since the treatment was instituted.

Fibrosis of the Esophagus; Cardiospasm.—Early in his studies of cardiospasm Mosher [73] became convinced that spasm played but a minor part in the cases observed by him. In a study of 100 autopsy specimens of the esophagus which were examined macroscopically as well as microscopically, it was shown that the esophagus, like all other organs of the

72. Moersch, H. J.: Further Studies on the Treatment of Esophageal Varices by Injection of Sclerosing Solution, Ann. Otol., Rhin. & Laryng. **50**:1233 (Dec.) 1941.

73. Mosher, H. P.: Infection as a Cause of Fibrosis of the Esophagus, Ann. Otol., Rhin & Laryng. **50**:633 (Sept.) 1941.

body, is subject to acute and chronic infection in acute and chronic systemic disease. It can be infected before birth, and it is also subject to extensive hemorrhage at birth. The most logical explanation for so-called spasm of the pylorus at birth is a slight fibrosis due to infection. In the 3 instances cited by Mosher, in which at birth there was a narrowing of both the pylorus and the cardiac end of the esophagus, this disorder is best explained by fibrosis due to antenatal infection rather than by double spasm.

Infection of the esophagus can originate within or without the esophagus or from the blood stream by an infectious thrombophlebitis. When the esophagus is infected from within, the pathway is probably the esophageal glands. It was shown by other writers many years ago that the superficial glands at the upper end of the esophagus ulcerate easily. Infection can reach these glands readily from the pharynx, a part of the body often subjected to infection, or from the apexes of the lungs. Below, the chief source of infection is disease of the liver and of the gallbladder. Autopsy specimens show that even infection of the appendix can be associated with infection of the esophagus. Periesophageal abscess is not uncommon, especially at the cardiac end of the esophagus. In the last 10 autopsy specimens of the esophagus recently examined 3 such abscesses were found.

Dilatation of the superficial veins of the esophagus is very common. Common also is hemorrhage about these veins. Mosher has found instances in which a single vein was greatly enlarged and thrombosed and was in close contact with the esophageal epithelium. One could easily see that any further enlargement of such a thrombosed vein would tend to push off the epithelium and expose the subepithelial tissue of the esophagus to infection. In the resting state the lower part of the esophagus is closed by the deep vertical folds characteristic of this locality. An ulcer caused by trauma or infection could easily glue these folds together and result in obstruction. One type of fibrosis of the terminal portion of the esophagus has this relatively slight pathologic condition as its cause. Such cases are most dramatic because the gluing is readily broken by instrumentation, and a quick cure of the patient results. Infection followed by fibrosis of connective tissue is an old pathologic entity and is a logical, proved and common cause of stricture of all grades in any part of the esophagus.

In the case of achalasia reported by Mitchell [74] the esophagus was tremendously dilated and contained about 2 liters of material. After removal of this a satisfactory view of the esophageal wall was secured. This was extremely thickened and granular. The diaphragmatic opening

74. Mitchell, H. E.: Achalasia of the Esophagus: Report of a Case Relieved by Esophago-Gastrostomy, Ann. Otol., Rhin. & Laryng. **50**:662 (Sept.) 1941.

could not be visualized owing apparently to deformity and fixation of the lower end of the esophagus. Since mechanical dilatation was not possible the patient was referred to the surgeon for esophagogastrostomy. This was carried out satisfactorily, and although the roentgenogram revealed that the defect was not entirely corrected the patient was able to eat without difficulty, gained weight and was clinically well.

Ball and Crump [75] reported a case of cardiospasm accompanied by a prolapse of the lower 10 or 12 cm. of the esophagus through the hiatus into the abdominal cavity. They attributed this unusual complication to the fact that the patient would force the esophageal contents into the stomach by swallowing air, taking a deep breath and attempting forced expiration with the mouth and glottis closed. No relief was obtained by forcible dilatation because of inability to pass the dilating instrument through the narrowed segment of the esophagus. To affect a cure esophagogastrostomy was performed.

In a study on the effects of avitaminosis B_1 on the motility of the bowel, Stinson [76] was impressed with the marked analogy between that and the finding of cardiospasm at autopsy. On the basis of this information he subjected several patients with cardiospasm to a clinical trial with thiamine chloride and found that there was a prompt response to treatment. He emphasized, however, that a patient with an obstructive lesion of the esophagus should not be treated with thiamine chloride until he had been subjected to esophagoscopy and an accurate diagnosis made. He directed attention to a case in which obstruction had been relieved for three months by the administration of thiamine chloride but later when an esophagoscopy was done it was found that this patient suffered from carcinoma.

Leiomyosarcoma of the Esophagus.—A fifth case of leiomyosarcoma of the esophagus was described in the literature by French and Garland,[77] in which the tumor was of the polypoid type with no metastasis or invasion of the deeper layers of the esophageal wall. A survey of the small group of reported cases shows that such a tumor may infiltrate extensively or remain a practically noninvasive polypoid structure. The authors stressed the point that a polypoid tumor may readily be mistaken for a mass of food, both on esophagoscopic and on roentgen examination. On esophagoscopy, at the junction of the middle and lower thirds a soft and friable mass was encountered which was mistaken for lumps of food until it began to bleed freely. In this case the biopsy specimen was

75. Ball, R. P., and Crump, A. C.: Mega-Esophagus (Cardiospasm), Radiology **36**:575 (May) 1941.

76. Stinson, W. D.: The Effect of Thiamin Chloride on Cardiospasm and Achalasia of the Esophagus, Ann. Otol., Rhin. & Laryng. **50**:898 (Sept.) 1941.

77. French, L. R., and Garland, L. H.: Leiomyosarcoma of the Esophagus, Am. J. Roentgenol. **45**:27 (Jan.) 1941.

reported as carcinoma, but subsequent postmortem studies revealed the true nature of the growth. Although bulky and partly ulcerated, the tumor was pedunculated and had not extended beyond the muscularis mucosae and might have been removable had the true pathologic condition been suspected.

Cancer of the Esophagus.—Although statistics show that 1.8 per cent of all deaths in the United States due to cancer are due to carcinoma of the esophagus, it is very probable that if the disease were diagnosed correctly in all cases the percentage would be much higher. Some observers have placed it as high as 8 per cent. According to Lindsay [78] failures by surgical treatment usually are due to the fact that the growth is very extensive. It is reasonable to assume, therefore, that appropriate treatment depends on early diagnosis, and this in turn can be secured only by a thorough examination of every patient with even mild discomfort, pain or difficulty in the act of swallowing. This includes not only a roentgen study but also early esophagoscopic examination.

Costen and Bryan [79] reviewed a series of 106 cases of esophageal carcinoma with special reference to diagnosis. In three fourths of the patients dysphagia constituted the initial symptom, with pain next in frequency followed by hoarseness and cough. Since the primary symptom of dysphagia is common to nearly all esophageal diseases the early diagnosis can be obtained only at the expense of routine complete examinations in all cases of unexplained dysphagia, pain in the chest, hoarseness or vomiting. Subjective symptoms of the mildest grade form the early basis of the diagnosis of carcinoma of the esophagus, and the first is disturbance in normal swallowing, long before actual difficulty is noted. The patient rarely consults a physician for such a complaint but notes its persistence, and only when the stage is reached that food "sticks in the throat" is the disorder considered worthy of report. The average survival rate of such patients was seven and six-tenths months; hence the short duration of this disease necessitates prompt action to insure the benefit of any form of treatment.

In discussing the possibilities of esophagoscopy in the diagnosis of disturbances in swallowing, Hagens [80] emphasized the responsibility that this procedure places on the endoscopist. An early positive diagnosis may make it possible to effect a cure by surgical treatment, whereas an indeterminate report may cause delay and as a result the disease may finally become too far advanced for surgical intervention. He pointed out the importance of securing a representative specimen of tissue for

78. Lindsay, J. R.: The Early Diagnosis of Carcinoma of the Esophagus, Ann. Otol., Rhin. & Laryng. **50**:675 (Sept.) 1941.

79. Costen, J. B., and Bryan, W. T. K.: The Diagnosis of Carcinoma of the Esophagus, Illinois M. J. **80**:274 (Oct.) 1941.

80. Hagens, E. W.: Carcinoma of the Esophagus: Endoscopic Experiences During the Past Year, Ann. Otol., Rhin. & Laryng. **50**:893 (Sept.) 1941.

biopsy. Often edematous tissue proximal to the growth may be removed, and this commonly does not contain cancer cells, with the result that the pathologist will not see any evidence of cancer. One must therefore employ a proper-sized tube, for a small esophagoscope often can be inserted through a strictured area and a more satisfactory view of the lesion itself can be secured. It also is important to observe the condition of the esophageal wall, namely, the presence of fixation and rigidity as well as stenosis.

While carcinoma of the esophagus has long been regarded as a hopeless disease with cure impossible and palliative treatment unsatisfactory, Benedict [81] directed attention to the facts that with early diagnosis radical surgical intervention may lead to cure and that even with the disease in advanced stages irradiation therapy combined with bougienage may give satisfastory palliative results. A case of a two year cure of carcinoma of the esophagus following radical resection was reported. In a series of 55 consecutive cases of carcinoma of the esophagus in which esophagoscopy was performed, 10 patients were treated surgically and the growth resected. It was interesting to note that in a number of cases the diagnosis of carcinoma was not suspected for a considerable time and in several the condition was considered as functional. Benedict therefore emphasized the importance of a roentgen study of the swallowing function and also esophagoscopy for all patients who exhibit symptoms referable to the esophagus. It is only by the establishment of the diagnosis early that an increasingly larger proportion of patients with carcinoma of the esophagus can be cured by radical operation. In the event that the lesion is too extensive to permit of surgical extirpation, much may be accomplished in the way of palliation by roentgen therapy combined in certain instances with bougienage. In his experience gastrostomy was seldom found necessary.

The apparent hopelessness of carcinoma of the esophagus has not aided greatly in early diagnosis. In discussing the surgical treatment, Johnson [82] pointed out that the difficulties in surgical technic have now been overcome to a great degree. With the advances in thoracic surgery during the past few years the operation for carcinoma of the esophagus is now sufficiently well standardized to insure a reasonably low mortality. Therefore, if further advances are to be made in the surgical treatment, the medical profession as well as the public must be taught that slight disturbances in swallowing should be investigated promptly. In Johnson's article a successful resection of the thoracic portion of the esophagus

81. Benedict, E. B.: Carcinoma of the Esophagus with Special Reference to Treatment by Radical Surgery, X-Ray and Bouginage, Ann. Otol., Rhin. & Laryng. **50**:1249 (Dec.) 1941.

82. Johnson, J.: The Surgical Treatment of Carcinoma of the Thoracic Esophagus: Case Report with Description of New Artificial Esophagus, Ann. Otol., Rhin. & Laryng. **50**:1228 (Dec.) 1941.

for carcinoma was reported, and a new type of artificial esophagus was described.

In a comprehensive review of the literature on the surgical treatment of carcinoma of the esophagus 195 cases were collected, which included 4 reported by Ochsner and DeBakey.[83] The relatively high and apparently increasing incidence of carcinoma of the esophagus, which constitutes approximately 5 per cent of all carcinomas, and its preponderance of occurrence in the thoracic portion of the esophagus, together with its obstinacy to all other forms of therapy, clearly emphasize the importance of surgical treatment. The various plans of surgical treatment were reviewed and critically analyzed. Four patients surgically treated by the authors were reported on. One of these was still living at the time of the report, over one and one-half years following operation.

The high morbidity and mortality rate of esophageal perforation is due in part to the esophagus being surrounded by a potential space of loose connective tissue which provides an extensive area for the rapid unobstructed advance of infection. In presenting the complications of esophageal perforations, Holinger[84] discussed the anatomic considerations, etiology, signs and symptoms and the general problems of treatment as well as specific complications of esophageal perforations. Seven cases are reported, and the end results are recorded.

The complications of esophageal perforations are enumerated as follows: periesophageal infections and abscess; hemorrhage; perforations of the pleura; emphysema; pericarditis, and perforations of the trachea and bronchi. Associated perforations of the pleura, trachea and bronchi or hemorrhage as a rule are readily recognized and treated according to well established principles. Mediastinal infections demand prompt drainage. This includes not only those cases in which there is definite evidence of a mediastinal abscess but also those cases of gross perforation demonstrated at endoscopy or by roentgenography.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES

Bailey[85] called attention to the too frequently overlooked fact that after aspiration of a foreign body there is nearly always a period free of symptoms accompanied by one important sign, a wheeze heard at the open mouth. It is important that the diagnosis of foreign body in the bronchus be made as soon as possible and before the sequelae of obstructive emphysema and atelectasis have occurred. During this

83. Ochsner, A., and DeBakey, M.: Surgical Aspects of Carcinoma of the Esophagus: Review of the Literature and Report of Four Cases, J. Thoracic Surg. **10**:401 (April) 1941.

84. Holinger, P. H.: Complications of Esophageal Perforations, Ann. Otol., Rhin. & Laryng. **50**:681 (Sept.) 1941.

85. Bailey, P.: Symptomless Period of Bronchial Foreign Bodies, Northwest Med. **40**:365 (Oct.) 1941.

symptomless period the significance of an occasional wheeze or cough should not be overlooked, in spite of an irrelevant history.

In a report of a series of 40 cases of vegetal foreign bodies which had been aspirated into the respiratory tract and had been removed by bronchoscopy, Richards [86] pointed out that in a statistical study of foreign bodies it was shown that nuts as foreign bodies compose about 32 per cent of the total number of bronchial foreign bodies. He also emphasized the frequency with which nuts enter the air passages in the form of nut candy. The youngest patient in his series was 10 months of age. A majority of the patients were under 4 years. The history is very important, and too often the physician first consulted pays little attention to it, with the result that the family is impressed that there is no foreign body present and often fail to recount the history of choking and gagging to subsequent physicians, so that the foreign body may be overlooked. The essential symptoms are fever, increased respiratory rate, cough and in some cases cyanosis and hoarseness. By far the greater number of patients show no acute illness. The three common signs observed on physical examination are unilateral limitation of expansion of the chest, hyperresonance of percussion, indicating the presence of obstructive emphysema, and greatly diminished or absent breath sounds. The question of anesthesia, the bronchoscopic observations, the operative procedures and the technical difficulties are discussed in detail.

There was but 1 fatality in Richards' series. A patient had been treated elsewhere as having pneumonia, and only after careful study of the history five weeks later was the suspicion of foreign body elicited (inhalation of a peanut). A portion of the nut fragment was removed from the right main bronchus. Believing that a fragment remained, the author searched further, but the patient suddenly ceased to breathe and there was cessation of the heart beat. It was believed that death was due to cardiac collapse, as the foreign body fragment did not seem to be sufficiently large to produce bilateral bronchial obstruction.

Observations on a series of 220 consecutive cases of foreign bodies in the air and food passages were made by McHenry.[87] One third of the patients were 3 years of age or less, while 60 per cent of the total number were in the first decade of life. Food accidents were responsible for foreign bodies of the air and food passages in 42 per cent of the total cases. Metallic objects accounted for over one third of the foreign bodies, while vegetable objects made up one fourth of the total. The great majority of the foreign bodies were removed within twelve hours after their lodgment. The eighteen safety pins included in this report

86. Richards, L. G.: Vegetal Foreign Bodies in the Bronchi: Analysis of Forty Cases, Ann. Otol., Rhin. & Laryng. **50**:860 (Sept.) 1941.

87. McHenry, L. C.: Foreign Bodies of the Air and Food Passages: Some Observations on a Series of Two Hundred and Twenty Cases, J. Oklahoma M. A. **34**:419 (Oct.) 1941.

were removed by one of four methods: (1) closure in the esophagus with the aid of the Mosher esophagoscope; (2) the point-sheathed method; (3) the dangling method; (4) straightening out of the pin. The author stated that the removal of some foreign bodies without relaxation afforded by general anesthesia carried in his hands a greater risk of serious complications than that arising from the anesthesia; consequently some form of general anesthesia was used in over half the operations. In this series of 220 consecutive patients there were 7 deaths, giving a mortality of 3.18 per cent. All but 1 of the patients that died was under 18 months of age. Three patients had mediastinitis, one of whom died. In the 2 who recovered surgical mediastinotomy was performed, although mediastinitis was present on admission. It was necessary to perform 15 tracheotomies, 3 of them before attempted removal of the foreign bodies. Besides asphyxia resulting from acute obstruction of the airway, suppurative pneumonitis and bronchiectasis occur after long-continued obstruction.

Three unusual cases of foreign body, in 2 in the air passages and in 1 in the stomach and esophagus, were presented by Bonnier [88] to emphasize certain points of clinical interest and methods of treatment. In a case of tracheal cannula in a bronchus there were no evidences of respiratory distress. The lower end of the cannula was in the right bronchus with the upper end resting on the carina. Removal was carried out with a forward grasping forceps passed through a 9 mm. bronchoscope. A case of safety pin in the bronchus of ten years' duration was reported. The outstanding symptom was slight hemoptysis. The pin had gravitated downward into the right lower lobe bronchus and had produced remarkably little local reaction. On removal it was fragmented because of corrosion of the metal. The patient made a prompt recovery. In the third case a corset bone had been swallowed; one end was in the stomach resting on the greater curvature while the upper end still remained in the esophagus. The ends were rounded and smooth, and the shaft was uniformly pliable. There were no evidences of trauma, and no difficulty was encountered in its removal.

The importance of accurate and timely roentgen examinations in all cases of suspected nonopaque foreign body in the respiratory tract was emphasized by Morrison and Walton.[89] The obstructive emphysema or atelectasis produced by such an accident must be diagnosed by roentgenograms made at the end of both inspiration and expiration together with the fluoroscopic examination. The roentgen examination should be repeated the following day or after a shorter interval if necessary in those cases in which there is a history of the aspiration of a foreign body

88. Bonnier, M.: Presentation of Three Interesting Endoscopic Foreign Body Cases, Ann. Otol., Rhin. & Laryng. **50**:936 (Sept.) 1941.

89. Morrison, A. E., and Walton, H. J.: Roentgen Diagnosis of Nonopaque Foreign Body in Lower Respiratory Tract, Am. J. Roentgenol. **45**:843 (June) 1941.

and the initial examination showed no evidence. Roentgen study of the size, shape, nature and probable site of lodgment of the foreign body is valuable to the bronchoscopist.

In a consideration of the problem of the open safety pin, Richards [90] asserted that familiarity with one dependable method is more valuable than a theoretic knowledge of several. Pins lodged in the food passageway appear in their full expanse on a true anteroposterior roentgenogram, while those in the airway present a similar picture only on the true lateral view. In his experience there are more advantages than disadvantages accruing from the use of a general anesthetic except in the presence of respiratory embarrassment; the resultant relaxation of the esophageal musculature is an advantage that far outweighs any objection to general anesthesia.

In the few cases in which the safety pin lies point down in the hypopharynx or esophagus it may be removed by grasping the spring with rotation or alligator forceps, or what may be termed "toggling," and extraction accomplished with the point trailing. A pin lodged in the hypopharynx in the point up position can be removed by one of two methods. In the first the keeper is grasped through the eye with a rotation forceps and the pin removed by dangling or toggling. In the second method a version is performed with the tip of the laryngoscope used as a fulcrum for the point on its inversion. For a pin in the cervical or thoracic parts of the esophagus, Richards recommends the use of the Mosher safety pin closer; if this method is not successful he advances the pin into the stomach and deposits it. At times it may be necessary to remove the foreign body from the stomach after a reasonable delay. A pin lodged in the larynx point up can be safely removed by grasping the keeper and traction exerted by toggling. If the point of the pin is engaged in the anterior glottic wall below the vocal cords, partial straightening of the pin through the larynx must be performed. A pin lodged in the bronchus is best removed by the straightening method, in the author's experience. Fluoroscopic aid is indispensable in the removal of pins in the cervical and thoracic parts of the esophagus and is a distinct aid with those in the tracheobronchial tree.

The case of foreign body in the esophagus reported by Friedberg [91] presented an unusual group of symptoms. The patient, a woman aged 35, complained of severe sudden pain in the chest which occurred while eating. She had no knowledge of having swallowed a foreign body. The history suggested a pneumonic process, embolism or cardiac dis-

90. Richards, L. G.: The Open Safety Pin: Consideration of Its Peroral Removal from Upper Air and Food Passages, New England J. Med. **225**:561 (Oct. 9) 1941.

91. Friedberg, S. A.: Esophageal Foreign Body with Symptoms Suggesting Other Thoracic Disease, Arch. Otolaryng. **34**:591 (Sept.) 1941.

turbance. Certain of the symptoms were suggestive of the presence of a foreign body in the esophagus, but there was absence of dysphagia and odynphagia and the roentgenograms all were normal in spite of the fact that barium sulfate had been given together with a small mass of cotton which it was believed would adhere to a foreign body if one was present. After the patient had been observed for a time and serious cardiovascular and pulmonary lesions had been ruled out it was decided to do an esophagoscopy. A large portion of flat bone was found in the esophagus in its middle third. Removal of this was followed by immediate relief from the severe pain in the chest.

A report of 2 cases of perforation of the esophagus by foreign bodies with recovery was made by Cummings.[92] In both cases there was recovery without mediastinotomy following the administration of sulfanilamide. A chicken bone had perforated through the piriform sinus in one of the cases, forming an abscess; after removal the opening was enlarged with forceps and drainage into the pharynx followed. In the other case it was thought the point of a safety pin had perforated the middle part of the esophagus; after removal of the pin recovery was uneventful. The author contended that immediate operation for esophageal perforation is not always necessary and that the condition of the patient together with changes in the physical and roentgenologic findings are of prime importance in determining the question of operation.

While foreign bodies which lodge in the cervical portion of the esophagus usually can be removed without much difficulty, an occasional case is observed which does not permit of esophagoscopic removal. Myerson [93] reported a case of lodgment of a beef bone in the upper part of the esophagus which could not be removed with three attempts at esophagoscopy. The bone, part of a vertebral unit, did not permit of grasping with forceps because of the hard sloping surfaces which were presented. There also was marked tissue reaction, so that no instrument could be passed between the foreign body and the esophageal wall. External esophagotomy was performed and the bone removed. At that time there were found marked necrotic changes with pus outside the esophagus. A blood stream infection ultimately developed, with thrombosis of the internal jugular vein, which was ligated and removed. Death resulted after several severe hemorrhages. At postmortem examination there were found a gangrenous esophagitis and periesophagitis, mediastinitis and multiple abscesses of both lungs.

A series of 93 cases of perforation of the intestinal tract by ingested foreign bodies has been collected from the literature and studied by

92. Cummings, G. O.: Perforation of Esophagus by Foreign Bodies: Report of Two Cases with Recoveries, J. Maine M. A. **32**:193 (Aug.) 1941.

93. Myerson, M. C.: Foreign Body in Upper Esophagus Requiring External Esophagotomy, Laryngoscope **51**:1059 (Nov.) 1941.

Macmanus [94] with the addition of 2 recent cases of his own. Seventy-three per cent of the perforations occurred in and around the ileocecal region, with the appendix the chief site, and the remainder were distributed generally from the stomach to the rectum. Six cases of perforation of the intestine with a hernial sac were reported. The duration of symptoms ranged from a few hours to two weeks in 50 per cent of the cases, while the remainder of the cases had longer histories. The types of symptoms were of two patterns: one, a sudden sharp mid-abdominal pain usually accompanied by vomiting which resulted from sudden perforation with immediate soiling and subsequent infection of the peritoneal cavity; the other, a more gradual onset of abdominal pain with low grade fever and localized tenderness heralding the development of an intraperitoneal inflammatory mass. The foreign bodies in this study were classified into three broad groups. The first group was composed of metallic bodies, under which were included pins of various descriptions and segments of wires; these comprised 46 per cent of the total. Animal bones, comprising fish bones and chicken bones, and also a wide variety of fragments of bony spicules, ranging from portions of a rabbit's rib to teeth, made up the second group of 45 per cent. Wood splinters, including toothpicks and in 1 case a portion of pencil, were responsible for the remainder of the perforations. The mortality from intestinal perforations was reduced in the last forty years from 53.5 to 10 per cent.

GASTROSCOPY

Gastroscopy as a valuable aid in diagnosis is recognized not only by gastroenterologists but by internists, surgeons, radiologists and others who are beginning to appreciate its importance. Levin and Shushan [95] discussed the established indications for gastroscopy and also emphasized that the field probably will eventually embrace a miscellaneous group of diseases which hereto have been incompletely studied. These include blood dyscrasias and food deficiency diseases, as well as certain infectious diseases. They emphasized that the procedure does not induce any special discomfort and in the hands of one properly trained is safe. They stated that in the examination of approximately 300 patients, some of them two or three times, there were no mishaps. A plan of procedure which is believed by them to be somewhat simpler than the original Schindler-Wolf method is reported.

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94. Macmanus, J. E.: Perforations of Intestine by Ingested Foreign Bodies: Report of Two Cases and Review of Literature, Am. J. Surg. **53**:393 (Sept.) 1941.

95. Levin, A. L., and Shushan, M.: Gastroscopy as a Diagnostic Procedure: A Clinical Study with Illustrative Cases, Rev. Gastroenterol. **8**:103 (March-April) 1941.

Browne and McHardy [96] contended that gastroscopy is a safe and most accurate method of immediately determining the source and character of gastric bleeding. They offered for consideration as possible indications for gastroscopy: acute melena and hematemesis free of "shock" or other contraindications, the management of which demands an immediate diagnosis. Gastritis may occur alone or concomitant to an ulcer, a malignant growth or a previous gastroenterostomy and as such may be the sole source of bleeding while the more obvious lesion is suspected. Small benign tumors, friable gastric mucosa, gastric purpura, mucosal erosions of hepatic insufficiency and other noninflammatory conditions are listed as sources of bleeding, the diagnosis of which is readily adaptable to gastroscopy. When emergency surgical intervention is contemplated gastroscopic investigation would prevent many hazardous invasions of the abdomen. In three years the authors have encountered only 12 patients with acute hemorrhage in whom they judged the procedure applicable, and of the 10 for whom laparotomy was considered only 4 were treated surgically.

The left-sided position is the one commonly employed in flexible tube gastroscopy. The common objections to the right-sided position are: secretions collect in the antrum and interfere with a satisfactory view of the pylorus, there is increased danger of injury at the cardia, there is a slight forward swinging of the body of the stomach which partially obstructs a satisfactory view and, in addition, the technic is awkward for the operator. In examining a patient who had dextrocardia and a transposition of the gastrointestinal tract, Berry [97] found that the right-sided position more nearly approximated the standard relations, since the stomach in this case was on the right side. He had no difficulty in passing the gastroscope into the lower depths of the stomach and the patient experienced practically no discomfort. Later gastroscopy was done with the patient in the standard left-sided position. It was possible to see practically all of the proximal lesser curvature of the antrum which formed the usual "blindspot." The "blindspot" on the posterior wall present in the reverse was entirely absent in this position. He therefore decided to try the right-sided position for examination of patients with normally placed stomachs. These were first examined in the standard left-sided position; then the gastroscope was withdrawn and reinserted with the patient in the right-sided position. He concluded that when lesions are suspected in areas which remain "blind" in this position an experienced operator who has familiarized himself with the

96. Browne, G. D., and McHardy, G.: Gastroscopy in Hemorrhage of Stomach, New Orleans M. & S. J. **93**:338 (Jan.) 1941.

97. Berry, L. H.: Right Side Gastroscopic Technique in Situs Inversus Viscerum and in Visualization of "Blind Spots," Rev. Gastroenterol. **8**:267 (July-Aug.) 1941.

reverse orientation may discover hidden lesions, as he was able to do in 6 of his patients.

Perforation of the stomach is said to occur very infrequently following the employment of the flexible gastroscope. Five perforations reported in a series of 4,000 examinations carried out independently by six leading gastroscopists resulted following the employment of the gastroscope with the "sponge tip." None of these terminated fatally. Schindler and others have recommended the employment of a long rubber finger instead of the sponge tip. They believe that the long finger, which directs the instrument in the line of least resistance and initiates bending of the flexible portion, distributes the pressure on the stomach surface. Schiff, Stevens and Goodman [98] reported the case of a perforation with a gastroscope equipped with a finger tip. Whether the pneumoperitoneum which developed was due to the instrument penetrating the stomach wall or to overdistention of the stomach with air could not of course be established. The stomach wall probably was thinner than normal because of atrophy of the mucosa. More pressure than usual was probably exerted on the posterior wall of the stomach near the greater curvature, as evidenced by the fact that the tip of the instrument was observed to be bent on itself. Curiously, the patient exhibited no untoward symptoms, and the pneumoperitoneum was discovered four days after the gastroscopic examination when the patient was sent to the roentgen ray department for a gastrointestinal series. Surgical intervention was not indicated, and the patient made a complete recovery.

An unusual case of perforation of the cervical portion of the esophagus with the flexible gastroscope was reported by Touroff.[99] The finger-like rubber bougie on the distal portion of the instrument perforated the posterolateral esophageal wall, became detached and remained lodged in the neck after withdrawal of the gastroscope. The patient was promptly operated on and the neck and mediastinum drained. The operation was followed by rapid recovery.

A case of perforation of the stomach by the flexible gastroscope was reported by Schiff and Shapiro.[100] A striking feature of this case was the absence of pain at the time of the accident. The site of the perforation was high on the posterior gastric wall, and recovery followed laparotomy.

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98. Schiff, L.; Stevens, R. J., and Goodman, S.: Pneumoperitoneum Following Use of Flexible Gastroscope, Ann. Int. Med. **14**:1283 (Jan.) 1941.

99. Touroff, A. S. W.: Perforation of Cervical Esophagus with Flexible Gastroscope: Case Report—Diagnosis—Treatment, Ann. Surg. **114**:369 (Sept.) 1941.

100. Schiff, L., and Shapiro, N.: Perforation of Stomach with Flexible Gastroscope: Case Report, Am. J. Digest. Dis. **8**:260 (July) 1941.

Abstracts from Current Literature

Ear

THE DIAGNOSTIC SIGNIFICANCE OF PARTIAL PARALYSIS OF THE FACIAL NERVE IN CHRONIC SUPPURATIVE OTITIS MEDIA. HAROLD I. LILLIE, Ann. Otol., Rhin. & Laryng. **50**:38 (March) 1941.

The author traces the course of the facial nerve from the nuclei to the distribution, and, on the basis of this, diagnoses the location of the lesion affecting the facial nerve according to the function involved. A cerebral lesion, because it causes paralysis of the upper neuron type, spares the occipitofrontalis, the orbicularis palpebrarum and the corrugator supercilii muscle. A lesion in the upper part of the pons causes involvement of the pyramidal tract on the same side. A lesion in the lower part of the pons produces involvement of the opposite side. If the lesion is in the medulla the eye on the same side should turn inward, owing to involvement of the sixth nerve, and there should be an accompanying effect on the pyramid and fillet. A lesion proximal to the geniculate ganglion usually causes impairment of hearing and equilibrium. Involvement of the geniculate ganglion (Ramsey Hunt syndrome) is indicated by herpes of the external auditory canal and the adjacent portion of the auricle. If the lesion is distal to the geniculate ganglion the paralysis is of the lower neuron type (flaccid) and involves all of the facial muscles. If sensation of taste is absent from the anterior two thirds of the tongue the lesion is somewhere between the geniculate ganglion and the point at which the chorda tympani nerve leaves the nerve trunk to pass through the middle ear.

Diagnostic points are cited from the literature, and several case reports are presented which seem to show that partial facial paralysis occurring before or soon after operation on the ear has diagnostic significance in localizing the lesion in the nerve pathway. Early operation for relief of compression or correction of the injury seems rational treatment. Partial facial paralysis occurring in the presence of chronic suppurative otitis does not necessarily mean that the lesion causing the paralysis is situated in the intracranial nuclei.

M. V. MILLER, Philadelphia.

AN EVALUATION OF THE MÉNIÈRE SYNDROME. W. E. GROVE, Ann. Otol., Rhin. & Laryng. **50**:55 (March) 1941.

This is a review of the facts as they have been shown by the many investigators regarding the etiology and pathology, together with the symptomatology and diagnosis, of Ménière's disease. Treatment is discussed in relation to the various theories of etiology. The author feels that patients suffering from this condition should be given the opportunity of trying medical treatment before submitting to surgical intervention.

M. V. MILLER, Philadelphia.

CLOSURE OF PERFORATIONS OF MEMBRANA TYMPANI WITH CARGILE'S MEMBRANE. W. D. STINSON, Ann. Otol., Rhin. & Laryng. **50**:178 (March) 1941.

While an intact ear drum is not essential to good hearing, it is always desirable and should be obtained if possible. With a perforation, localization of sound is interfered with to some extent, danger of infection is always present and certain limitation of sports, such as swimming, is necessary. A traumatic perforation tends to close spontaneously, and even an old perforation frequently closes after acute otitis media. Stinson feels that a drum membrane will always close provided the inflammatory condition in the middle ear subsides, unless union occurs

between the epidermal and the mucosal layer of the drum membrane. He points out that the epidermis of the drum membrane, in the tense portion, migrates on its original plane from the anterior margin of the membrane across to the posterior canal wall and then along the wall of the canal to the external meatus. In the flaccid portion it fans out over the wall of the canal. The proliferation about the short process is likely to proceed in whorls, but the movement tends to be toward the superior rather than toward the posterior part of the wall. The rate of progress is not uniform but is more rapid in the posterior inferior quadrant.

In attempting to close these perforations, Stinson uses sheep mesentery, marketed by supply houses as Cargile's membrane and by drugstores as fishskin condom. Stinson prefers the latter, as it is readily available and is thinner. Before making the attempt the middle ear must show no signs of inflammation; the edges of the perforation, if it is several years old, are touched with trichloroacetic acid and one or two days is allowed for the inflammation incident to the application to subside. The external auditory canal is carefully cleansed, and any hairs projecting into the canal are trimmed to prevent the membrane from adhering to them. The size of the perforation is estimated, and the size of the membrane to be used must be at least 25 per cent larger than the perforation to be closed. A small wisp of cotton is wrapped on a metal applicator and touched to the center of the membrane. Care must be taken that the membrane is exactly centered over the perforation before it touches the drum membrane, as it seems to leap into place as soon as it does so. Healing takes place in from ten to thirty days.

M. V. Miller, Philadelphia.

Cochlear Activity After Death. E. G. Wever and Merle Lawrence, Ann. Otol., Rhin. & Laryng. **50**:317 (June) 1941.

The authors report their investigations on the death function in the cochlear response, considered with respect to the cause of death, the conditions of stimulation and the concomitant physiologic changes. The animals were killed by pithing the medulla, clamping the heart, clamping the trachea or poisoning with curare. All of these methods produce prompt impairment of response, with the most rapid loss caused by clamping the heart and the most moderate initial effect by curare. After a large initial loss the responses decline relatively slowly and occasionally show periods of stability and partial recovery. Responses are still present after five hours but not after twenty-four hours. There is apt to be a "rebound" immediately after the initial drop. It occurs in the absence of the middle ear and so is not caused by relaxation of the tympanic muscles. The same is true of the later periods of partial recovery. No simple relation has been observed between the cochlear response and changes in blood pressure. The initial loss of response seems unrelated to body temperature, but there may be a relation to the later course, as even slight applications of heat cause a noticeable increase in the responses. All frequencies suffer the initial drop, but curves for different tones follow slightly different courses. Usually the high tones show the most rapid decline, but exceptions occur. Death reduces the magnitude of responses without producing any significant variation in the form of the intensity function. The course of the change is practically independent of the degree of stimulation of the ear. The authors feel that these facts indicate that in death the cellular changes are simple and that there is no alteration of the effective pattern of stimulation. They suggest that the cochlear potentials arise through direct translation of stimulus energy and not through liberation of stored energy.

M. V. Miller, Philadelphia.

The Ear in Experimental Vitamin A Deficiency. H. B. Perlman and John Willard, Ann. Otol., Rhin. & Laryng. **50**:349 (June) 1941.

The authors report the result of their experiments on the effect of vitamin A deficiency on the ears of rabbits. As an index of cochlear function they used the acoustic reflex contraction of the tensor tympani muscle, testing the animals under

sterile precautions before the start of the experiment and again at the end. The diet was carefully watched to exclude all vitamin A and its carotenoid precursors. Rabbits about 8 weeks old were used and were kept on the diet for from four to eight months. A number of control animals were followed in the same manner. It was found that there developed extensive hypertrophy of the periosteal layer of the otic capsule on the cranial aspect, including the internal auditory meatus, the surface of the posterior fossa and the mouth of the flocculus. The overgrowth of bone in the internal auditory meatus produces narrowing and elongation of the canal, with compression and stretching of the nerve fibers and Scarpa's ganglion. In spite of this compression and stretching good cochlear function was retained, but there did result various degrees of degenerative changes in some of the cells in the vestibular and cochlear ganglia, and in 1 case degeneration of the peripheral fibers in part of the basal coil. There was not evidence of serous labyrinthitis or of degeneration of Corti's organ, the vestibular end organs or the stria vascularis. The structures of the middle ear, including the adjacent labyrinth bone, were unaffected.

M. V. MILLER, Philadelphia.

THE REACTIONS IN THE EAR TO SOUND. A. G. POHLMAN, Ann. Otol., Rhin. & Laryng. **50**:362 (June) 1941.

Pohlman discusses at length the theories as to how sound waves affect the fluid in the internal ear—whether they reach there through the structures of the middle ear or through bone. He assumes that the auditory cells are specific in their reactions to the frequencies of sound vibrations and that they are directly activated by the pulsating pressures applied to them through the labyrinthine fluid. He also assumes that the anatomy of the apparatus may be correlated with a shock-absorbing function, which protects the end organ against mass displacements in the cochlear liquid. The evidence seems to indicate that while the condition of sound vibrations to the auditory cells presents a simple problem in physics, the frequency analysis is dependent on a physiologic reaction of which little or nothing is known. He feels that there is no indication that much can be done to relieve perceptive deafness but that more may be accomplished for conduction deafness if scientific investigation is continued.

M. V. MILLER, Philadelphia.

OTOSCLEROSIS: SYMPTOMS IN THE EXTERNAL AND MIDDLE EAR. GUNNAR HOLMGREN, Acta oto-laryng. **29**:91, 1941.

So much attention has been focused on the labyrinthine capsule and the inner ear that the majority of textbooks do not even mention symptoms in the external canal and middle ear.

Nevertheless, in otosclerosis great and usually easily discernible changes are present in the external auditory canal, the tympanic membrane and the chain of ossicles. As early as 1860 Toynbee found that cerumen was absent in the external canal in advanced cases of otosclerosis. This has been confirmed by numerous authors.

The reduced cutaneous sensitivity of the external canal is also well known. The membrana tympani is more or less anesthetic, and the irritability of the canal to tickling is markedly reduced.

It was established in 1890 by Gourivand that the canal is wide and by Jorgen Moller in 1911 that it is wide and exhibits atrophy of the skin. This atrophy of the skin has been confirmed by others, notably by Leesen in 1928.

In regard to the appearance of the membrana tympani descriptions vary from "retracted, opaque, thickened" to "abnormally transparent." Bezold, Zwaidemaaker, Bonnier and Lemos observed restricted motion in the ossicular chain, but for this fixation of the stapes was considered to afford a satisfactory explanation.

One can study the motility of the ossicles with a Siegel otoscope and see movement in the manubrium and in the membrana tympani during aspiration and compression. One can estimate the amount of movement of the manubrium only in

relation to other "fixed" structures. But the wall of the external canal is not fixed, and this makes the estimation more difficult. This difficulty can be overcome if two parallel hairs set on either side of the manubrium are inserted into the Siegel otoscope. By the use of this procedure in otosclerosis it is found that the manubrium is often immobile, still more often shows restricted motility and relatively seldom exhibits normal motility. In a study of cases of otosclerosis an attempt was made to exclude all which showed any signs of past inflammatory changes in the middle ear. Holmgren thus considers it established that in numerous cases of otosclerosis the ossicular chain is ankylosed.

A couple of years ago Covell and Nager went through Nager's large collection of histologic otosclerotic specimens. Their study involved 46 temporal bones with otosclerotic foci in the region of the stapediovestibular joint, 11 in which otosclerotic foci were located elsewhere and 21 normal temporal bones for control. It proved that a disturbance in the position of the ossicles could be demonstrated in 95 per cent of the cases in which there were otosclerotic foci in the region of the stapes joint but not in a single case in which the focus was elsewhere; that the dislocation was most pronounced when a mantle was present near the malleoincudal joint—in 56 per cent and 86 per cent, respectively, of the two groups of cases—and that atrophy of the tensor tympani muscle was present in the severest cases of ankylosis. Covell, therefore, like Holmgren, considers the stapes fixation responsible for the rigidity of the other articulations.

In many cases of otosclerosis the thin and abnormally transparent pars tensa is much more mobile than normal, sometimes flapping like an atrophic tympanic membrane.

The vascular reflex observed in normal persons in the production of hyperemia of the membrana tympani and the skin of the superior-posterior wall of the auditory canal when the canal is stimulated by a cotton swab, aspiration or suction by a Siegel otoscope or the projection into the canal of a strong light (heat effect) is either absent or greatly reduced in cases of otosclerosis. The cough reflex from the external auditory canal is also greatly reduced.

Therefore, the symptoms of otosclerosis relating to the external auditory canal and middle ear are: reduction or absence of cerumen; reduction of sensibility to tickling; widening of the lumen of the external auditory canal, possibly due to atrophy of its soft parts; occasionally, abnormal transparency and thinness of the tympanic membrane, and, in numerous cases, rigidity and even ankylosis of the ossicular chain (histologically confirmed by Covell).

Because of the rigidity of the chain of ossicles, the umbo becomes fixed and the movements of the tympanum restricted, and because of such restricted movement there is deterioration in the nutrition of the tympanic membrane, which becomes thin, transparent and more mobile than usual.

The deterioration in circulation secondarily affects the vessels and nerves of the external auditory canal, where atrophy sets in and, with the atrophy, reduced sensitivity of the nerves. The trigeminus (sensitivity to tickling), the vagus (cough reflex), the vessel nerves (reflex hyperemia) and the secretory nerves (production of cerumen) are affected.

GROVE, Milwaukee.

Larynx

MANAGEMENT OF CLINICAL PROBLEMS INVOLVING THE LARYNX IN INFANCY AND CHILDHOOD. CLYDE A. HEATLY, New York State J. Med. **41**:607 (March 15) 1941.

Because of its anatomic structure, the infantile larynx is much more likely to collapse and cause asphyxiation than is that of older children. Heretofore the physician has "guessed" that the obstruction was "just a cold," but now he should discard the "inferential" for the differential method of direct laryngoscopy. Nondiphtheritic causes are hard to differentiate clinically from diphtheritic ones in

many cases. One cannot afford to wait for a report on a culture, but it should always be taken, nevertheless. Prompt tracheotomy may save a life, particularly in cases of laryngotracheobronchitis, with its tough, sticky secretion. Heatly speaks of the "chronic larynx," as evidenced by continued stridor, hoarseness or dyspnea. Analysis of causes in 20 cases shows: congenital laryngeal stridor, 12 cases; paralysis of the vocal cords, 2 cases; tumor of the larynx, 1 case; congenital web, 1 case; subglottic tumor, 1 case; subglottic stenosis, 1 case; tumor of the mediastinum, 1 case, and congenital anomaly of the ligamentous attachment of the sternum, 1 case. The commonest tumor seen in infancy is papilloma—congenital, multiple or recurrent. Angiomas and cysts occur now and then.

Heatly says that both the acute and the chronic disorders of the larynx in infancy present many difficult problems. These may be simplified by making a direct inspection in every case, taking material for culture, removing secretions or other obstructions and making a differential instead of an inferential diagnosis, thus leaving nothing to chance.

VOORHEES, New York.

TOTAL LARYNGECTOMY. H. CAVE and M. ESCAT, Oto-rhino-laryng. internat. **25**:49 (April) 1941.

The two chief objections to total laryngectomy in treatment of cancer of the larynx, the gravity of the operation and the crippling of function, are unjustified. The Glück-Tapia technic has resulted in a mortality rate of 10 per cent and in ultimate arrest in 50 per cent of the cases of extrinsic cancer. The crippling of function may be largely compensated by the development of a pharyngoesophageal voice or by the use of a perfected artificial larynx. The superiority of surgical measures over physical therapy has been amply proved. It is only in cases in which the outcome is in doubt, owing to the extent of the lesion or to the poor general condition of the patient, that other than surgical means may be considered.

In general, total laryngectomy is indicated in all cases in which the extension of the tumor renders uncertain its complete removal by partial laryngectomy, provided that the absence of lymphatic, pharyngeal and cutaneous involvement warrants the opinion that ablation will be complete. The operation is indicated in cases of cancer of the vocal cord when the cord and the arytenoid cartilage are fixed, when the arytenoid cartilage is involved or when the anterior commissure is widely crossed; in cases of cancer of the ventricular band when the base of the epiglottis is invaded, when the hemilarynx is immobilized or when the tumor tends to saddle the anterior commissure; in cases of cancer of the anterior commissure when the tumor has extended so far backward as not to be removable by anterior, frontal hemilaryngectomy, and in cases of subglottic cancer when the tumor approaches the middle line in front or in back. Laryngectomy is contraindicated (*a*) when the extension of the tumor to the pharynx is such that too great a pharyngeal resection is required to permit closure of the pharyngeal breach, (*b*) when there is tracheal or cutaneous involvement and (*c*) when cancerous adenopathy is present.

The chief advantages of the Glück-Tapia technic are the avoidance of preliminary tracheotomy, dissection of the larynx from above downward and employment of local anesthesia exclusively. Of supreme importance is the preoperative and postoperative care. Especially important is the use of insulin therapy if there is hyperglycemia, in order to bring the blood sugar content to normal. It is necessary to separate the dressing of the tracheal wound from the cervical dressing to prevent the complication of bronchopulmonary infection, and even to resort to two or more changes of dressing daily. The nasal feeding tube is removed between the tenth and the fifteenth day.

Seven illustrative cases are recorded.

DENNIS, San Diego, Calif.

Nose

OSTEOMA OF THE FRONTAL SINUS. GEORGE M. COATES and FREDERICK KRAUSS, Ann. Otol. & Laryng. **50**:450 (June) 1941.

Osteoma is more frequently in the frontal sinuses than in any of the others. The tumor may arise from the anterior or the posterior wall, but more commonly the former, and is more often pedunculated than sessile. It may be hard or soft, the former type being of dense compact bone while the latter is softer, even approaching the cancellous type. There are intermediate types also. Usually of slow growth and causing no symptoms in the early stages, the tumor is often found by roentgenographic examination or during operation. Infection of the frontal sinus may be caused by the growth if its location blocks drainage and prevents aeration. Gradual development of headache due to gradual increase in pressure is often the first sign, but headache may not appear until the tumor presses on an adjacent wall or structure. If growth extends inward the dural plate may be absorbed, but more often extension is downward or outward, resulting in deformity over the eyebrow or displacement of the orbit and limitation of its motion. Diagnosis rests on the external deformity, which may be the only symptom, pain, invasion of the orbit or brain cavity and roentgenographic evidence. The only confusion likely to occur is in the differential diagnosis of osteoma and the osteogenesis described by Skillern. Growth is usually gradual and progressive but may become stationary. Treatment is entirely surgical as soon as symptoms warrant removal. Two cases are reported.

M. V. MILLER, Philadelphia.

THE BACTERIOLOGIC RELATIONSHIP OF DENTAL INFECTION AND CHRONIC HYPERPLASTIC SINUSITIS. R. CLARK GROVE and JOHN G. WELCH, Laryngoscope **51**:263 (March) 1941.

Grove and Welch made a bacteriologic study of 160 teeth extracted from 100 patients suffering from allergic manifestations, chiefly asthma. Hyperplastic sinusitis was present in 60 per cent of the patients; 92 of the 160 extracted teeth were vital and 68 were nonvital. Cultures were made of the root tips of 110 of the extracted teeth. Immediately after extraction each tooth was sterilized, after which the root tips were removed aseptically, ground up and cultured. The hemolytic streptococcus was recovered from 10 per cent of the root tip cultures, Streptococcus viridans from 80 per cent, the staphylococcus from 32 per cent and the pneumococcus from 5 per cent. More than one organism was frequently recovered from a culture.

These results were compared with Grove's previously published bacteriologic study of 108 hyperplastic antrum membranes removed at operation, in which the hemolytic streptococcus was present in 25 per cent, Str. viridans in 43 per cent, the staphylococcus in 57.5 per cent and the pneumococcus in 39 per cent.

The authors concluded that the bacterial nature of dental infections has little relation to that of chronic hyperplastic sinusitis.

WOOD, Newark, N. J.

THE VALUE OF CONSERVATIVE MANAGEMENT IN RHINOGENIC ORBITAL COMPLICATIONS. H. MELLER, Monatschr. f. Ohrenh. **75**:132 (March) 1941.

Of 72 patients observed at the Vienna Clinic with orbital complications caused by sinusitis, 50, or 70 per cent, were cured by conservative treatment. The presence of danger signals, such as an orbital phlegmon, cerebral symptoms, a septic picture or a disturbance of the fundus was accepted as an indication for more active intervention, such as opening of the sinuses, but in general a very good chance for success with conservative management was noted. Only in cases in which the orbital symptoms had not improved after the institution of conservative measures or in which the condition of the patient had been downgrade, was

surgical intervention indicated. The various methods of treatment are enumerated, from major extranasal or intranasal operations to luxation or clipping of the middle turbinate and the conservative ways of shrinking the tissues of the nasal passage with application of cocaine-epinephrine, heliotherapy, application of wet dressings and injections of calcium. There is little doubt that the shrinkage of the nasal mucosa is one of the major factors in the achievement of good results in the conservative treatment. The release of the pressure on the mucosa by retained pus gives the membrane a better chance to recuperate. A subsequent edema or inflammatory reaction which has followed the perforating vessels into the orbit will subside, especially if the bone itself has not as yet become affected. In some cases no pus will be encountered (in 5 of the reported cases) in spite of all symptoms pointing toward purulent sinusitis. This can be explained by previous evacuation into the nose or by perforation into the orbit. A charted summary of all 72 cases on which this report is based is appended.

The recorded material includes the sex of the patient (the majority were males), the age (64 per cent were in the first decade of life), the temperature on admission, the rhinologic findings, the roentgen findings, the orbital symptoms and their duration following the institution of therapy and the type of therapy employed in each case.

LEDERER, Chicago.

Miscellaneous

THE LOCAL USE OF SULFANILAMIDE POWDER AND HYDROGEN PEROXIDE IN WOUND INFECTIONS. L. SCHNEIDER, Am. J. M. Sc. **201**:208 (Feb.) 1941.

Schneider suggests that the local use of sulfanilamide powder in wound infections be preceded by thorough flushing of the wound with hydrogen peroxide and that the powdered sulfanilamide then be introduced into the wound in the form of a suspension in hydrogen peroxide. This is based on recent work demonstrating that increased oxygen availability increases the bacteriostatic action of sulfanilamide. Hydrogen peroxide is used as the oxidizing agent of choice because it is suspected of being the intermediate agent responsible for the toxicity of sulfanilamide to bacteria. Some recent unpublished experiments in vitro by C. L. Fox Jr. are mentioned. Fox found that the bacteriostasis produced by the addition of small amounts of hydrogen peroxide to cultures of Streptocccus haemolyticus containing 10 mg. of sulfanilamide per hundred cubic centimeters of medium was more than ten times that produced by corresponding amounts of sulfanilamide used alone and more than two hundred times that produced by equal quantities of hydrogen peroxide used alone.

The authors place in the wound at one time the total amount of sulfanilamide which usually would be given by mouth in twenty-four hours, and this is replenished daily. Because of the high concentration of sulfanilamide in the blood resulting from such procedure (on one occasion the blood determination sixteen hours after the local administration of 6 Gm. of powdered sulfanilamide showed a concentration of 5.4 mg. of the free drug and 10.2 mg. of the total drug per hundred cubic centimeters) all of the toxic manifestations produced by oral administration may also be expected. The author cites 2 cases in which serious wound infection with Str. haemolyticus and Staphylococcus aureus haemolyticus, respectively, was treated successfully by repeated local administration of powdered sulfanilamide and hydrogen peroxide.

The author suggests that zinc peroxide and sulfanilamide used locally might prove efficacious in the treatment of chronic undermining ulcers as a modification of the treatment advised by Meleney and Harvey, who used zinc peroxide applied locally and sulfanilamide administered by mouth.

SHAMBAUGH JR. and JENNES, Chicago.

THE DISTRIBUTION OF SULFANILAMIDE BETWEEN BLOOD AND CEREBROSPINAL FLUID WITH SPECIAL REFERENCE TO INTRASPINAL TREATMENT. S. KATZENELBOGEN, B. A. CRUVANT and C. SILVERBERG, Am. J. M. Sc. **201**:830 (May) 1941.

Katzenelbogen, Cruvant and Silverberg carried out studies on 31 normal patients in whom the cerebrospinal fluid was normal. For the determination of sulfanilamide in the blood and cerebrospinal fluid the method of Bratton and Marshall was used. Sixteen schizophrenic patients with no physical disease received by mouth therapeutic doses of sulfanilamide, namely, 25 grains (1.625 Gm.) every six hours, plus 15 grains (0.975 Gm.) of sodium bicarbonate, or a total of 100 grains (6.5 Gm.) in twenty-four hours. The medication was discontinued at the end of the third day. The permeability quotients varied from 1.06 to 1.68 at the end of the first day of medication, from 1.17 to 1.44 at the end of the second day and from 1.19 to 1.36 at the end of the third day. For 10 patients (5 psychotic patients with general arteriosclerosis and 5 psychotic patients with organic disease) who received single doses (2 or 5 Gm. of sulfanilamide) the permeability quotients ranged between 1.33 and 2.92 four hours after administration of the drug and between 0.71 and 1.23 twenty-eight hours after the drug was given. The authors concluded that oral administration of sulfanilamide assures the penetration of the drug into the cerebrospinal fluid in concentrations sufficiently high to render unnecessary intraspinal administration.

MICHAELS, Boston. [ARCH. NEUROL. & PSYCHIAT.]

RECONSTRUCTION OF FACIAL DEFECTS WITH LATEX COMPOUND: RESULTS RECENTLY OBTAINED. A. H. BULBULIAN, Proc. Staff Meet., Mayo Clin. **16**:313 (May 14) 1941.

The defects most commonly remedied with prostheses are nasal and aural defects. Among nasal defects, malignant growth perhaps plays the most important role, whereas trauma is more common among aural defects. A nasal deformity causes more suffering and embarrassment to the patient than any other type of deformity about the face, because loss of even a portion of the face produces a distinct change in one's appearance. Consequently, proper reconstruction of such a defect will not only bring about a remarkable alteration in the appearance of the person but often will change his or her mental outlook.

Prosthetic restoration with latex has been highly satisfactory in the author's experience. Latex is employed for temporary appliances to be used in patients awaiting subsequent plastic surgical repair, such as those who have undergone removal of a malignant growth, and also in some patients for whom a plastic operation is for some reason, such as age or the state of the tissues, not advisable.

H. MILLER, Philadelphia.

RADIATION DAMAGE TO TISSUE AND ITS REPAIR. E. M. DALAND, Surg., Gynec. & Obst. **72**:372 (Feb.) 1941.

Late changes in an irradiated field include telangiectasis of the skin, followed by hyperkeratosis, alopecia, fissures, fibrosis and later loss of the subcutaneous fat, marked endarteritis of the smaller vessels and degenerative changes in the fascia. The most serious of all complications is the development of carcinoma of the skin in the treated area. There is an incubation period between the use of roentgen rays and the development of cancer that ranges from three to eighteen years.

Numerous cases of grave change occurring in roentgenologists and in patients are listed. The surgical management consisted in broad excision of the involved skin followed by the use of either a Thiersch graft or a pedicle flap.

H. A. MILLER, Philadelphia.

Society Transactions

MONTREAL MEDICO-CHIRURGICAL SOCIETY, SECTION OF OTOLARYNGOLOGY

D. H. Ballon, M.D., *President*

E. A. Stuart, M.D., *Secretary and Abstract Editor*

Feb. 20, 1941

Clinical Aspects of Chemotherapy. Dr. Walter Scriver.

It is interesting that such an advantage as is given by sulfanilamide and its derivatives came out of international research. Compounds of this group were first used in Germany in experiments with mice, and the experimenter, Gerhard Domagk, was awarded a Nobel prize, which his government would not allow him to accept. Then they were used in France and next in England, where they were first applied clinically in the treatment of puerperal septicemia and proved extremely valuable. Next they came to North America and were investigated particularly at Johns Hopkins Hospital, Baltimore.

It is also interesting to hear about the many individual drugs in this broad group. Sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine), which caused so much furor, was synthesized in England. Sulfanilamide was rather unfortunately prepared in an elixir form by one manufacturer in the United States, and this elixir caused a number of deaths. It was the substances that were added and not the sulfanilamide that did the harm. However, this unfortunate experience led to the passage of the Food, Drug and Cosmetic Act of 1938. It also led to greater caution in the use of new drugs by physicians and to an increase in manufacturers' submissions of new compounds to the Council on Pharmacy and Chemistry of the American Medical Association, the body which considers therapeutic agents to determine whether they are suitable for inclusion in New and Nonofficial Remedies. It was perhaps because of these developments that the use of sulfapyridine was delayed in that country. Two years ago at the meeting of the American College of Physicians there was not a paper on the use of sulfapyridine, whereas in the Royal Victoria Hospital very good results had been obtained from its use in 100 cases of pneumonia.

Sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) was then brought out, and the background just outlined must be taken into consideration when one reads the papers on that compound. News is printed in the newspapers first and later in the journals. The value of sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine), one of the newest of the group, has yet to be proved. Why are there so many of these drugs? Investigators are attempting to get one that will kill the bacteria without killing the patient.

Speaking as a physician, I find it interesting to discuss the situation from the standpoint of what preparation it is desirable to give, the size of the dose, the frequency of administration, etc. First, the drug can be given orally, with the hope that it will be absorbed; obviously it will not be if the patient is vomiting. Then, it can be given intravenously or intramuscularly in soluble form, and it is even being applied successfully in ointment form. Very little sulfanilamide but about 25 per cent of the sulfapyridine administered may be excreted in the feces. In the liver it is conjugated as the acetyl salt. The acetylated drug has no effect on bacteria; so obviously it is the so-called "free drug" that remains in the blood that counts. Sulfapyridine in single oral doses of about 1 Gm. reaches its maximum concentration in the blood in about four hours. Sulfathiazole reaches its maximum level more rapidly and is excreted faster. It is completely eliminated in sixteen

hours. The optimum level in the blood is 4 to 15 mg. per hundred cubic centimeters. Tissue fluid concentration is 75 to 90 per cent of the blood level, and the spinal fluid level is 50 to 75 per cent of the blood level. Sulfathiazole is not good in cases of meningitis, as its concentration in spinal fluid is low. These drugs are excreted so rapidly that clinical use has determined that they must be given every four hours, the patient even being wakened during the night for doses, in order to keep the concentration up. Conjugation may affect the original dosage and the rate of excretion. To get into the tissues the drug must diffuse. Dr. Graham Ross has given me a rule of thumb which is useful for dosage for children. He gives 1 grain (0.065 Gm.) per pound (0.5 Kg.) of body weight per twenty-four hours. With this one can control the curve and get a concentration of 4 mg. per hundred cubic centimeters in the blood. Sulfanilamide comes in 5 and 7½ grain (0.32 and 0.5 Gm.) tablets and sulfapyridine in 7½ grain tablets, and there are powder forms for children so that smaller doses may be given.

To discuss the question in general from the standpoint of toxic effects, I think it is most important to realize that here are drugs which can be bought over the counter. They are too freely used, and many patients get into trouble with them. The Medico-Chirurgical Society has petitioned the legislature to make these drugs unavailable except on prescription, and I think all physicians should be behind this petition.

The first thing about the toxic effect is to forget the original idea that sulfates should not be used with these drugs. It used to be thought that not even magnesium sulfate should be used. This has been disproved; it does not increase the toxic effects. It used to be believed that these drugs produced acidosis. This again is not so, and therefore there is no need to give sodium bicarbonate. The vomiting that may occur is from stimulation of the vomiting center. The newest idea is to give vitamins and nicotinic acid along with the sulfanilamide compounds; I think this is an outgrowth of the use of nicotinic acid in high voltage roentgen ray therapy, with which it has been used to stop the nausea.

To discuss briefly the toxic symptoms, vomiting, of course, is the one that I think causes most concern, and sulfapyridine is the big offender. Sulfanilamide may produce vomiting, but not a great deal. Sulfapyridine causes vomiting in 60 per cent of cases and sulfathiazole in 40 per cent of cases. Fever may be a symptom, as in the case Dr. Hutchison presented, in which the temperature rose to 106 F. If the patient has been under chemotherapy and fever continues, it may be a drug fever. The compounds are all offenders in this respect, and just about equally, sulfanilamide possibly being the worst. Often the proof lies in discontinuing administration of the drug and having the fever disappear. Cyanosis is probably caused only by sulfanilamide and is due to the formation of aniline pigment. If the patient is exposed to bright sunlight or other light, cyanosis appears more often, but it does not do any harm. It will pass off quickly when fluids are forced and the drug therapy is stopped. Rashes are more common with sulfapyridine and sulfathiazole. These may resemble any of the exanthems and can be cured by forcing fluids and stopping the use of the drug. Next to the vomiting, depression is the worst symptom. It seems to be more severe with sulfapyridine but also occurs with sulfanilamide. I have encountered some patients with this toxic symptom, one child in particular, who came to present quite a behavior problem. Sulfanilamide causes a drop in the hemoglobin and often in the leukocytes. As regards headache, sulfanilamide is the worst offender. It also may cause hematuria, due to the slight local irritation of crystals excreted; sulfapyridine, of course, is much more likely to do this. Fortunately, by forcing fluids one can easily prevent this complication.

The only other things I wish to review are the uses of this chemotherapy. In the first place, it is really specific for erysipelas. The course of the disease is now only about forty-eight hours at the most, instead of five days or longer as it used to be. I once saw a woman weighing about 300 pounds (136 Kg.) who had a chill, a temperature of 104 F. and a pulse rate of 120. I thought she had pneumonia and started giving sulfapyridine. The next day I went to see her, and she

met me at the door, completely cured. The family said: "Don't you think you had better look at mother's leg?" And there was the answer. She had had erysipelas and not pneumonia.

For pneumonia the curative value is about the same with sulfapyridine and with sulfathiazole. If there are meningeal symptoms, sulfathiazole should not be used. If no demonstrable effect is obtained within seventy-two hours, there is not likely to be one. For the meningococcic infections sulfanilamide or sulfapyridine should be used. For infections due to the gonococcus and hemolytic streptococcus, sulfathiazole is useful, and also for gas gangrene. The value of sulfathiazole is still uncertain against staphylococcic infection. I think that as time goes on it will be found to be not as specific for this infection as for some of the others.

There will be new compounds brought out, for as yet the perfect drug has not been found. Sulfapyridine might be good, but it causes vomiting, as does sulfathiazole, which also does not diffuse in the spinal fluid. Sulfanilamide does not touch the pneumococcus. Some day the theoretically and practically perfect drug of this group may be discovered, but that day is not here yet.

Hematologic Aspects of Chemotherapy. DR. T. R. WAUGH.

In order to give one the proper perspective on this subject, it would seem advisable for me to go back a bit and review the pertinent ideas which have been held in hematology before these new drugs appeared.

The bone marrow produces the red blood cells, certain of the white blood cells, particularly the polymorphonuclear elements, and the platelets. For a long time a group of conditions have been recognized that are characterized by a diminution in the circulating blood of one, two or all of these different morphologic forms. This diminution has been attributed to a paralytic effect exerted in some way or other on the bone marrow, and therefore these changes in the blood have been referred to as myelotoxic conditions. For some of them the toxic agent has been known, while for others it has never been identified. Peculiarly, the paralytic effect sometimes confines itself to inhibition in red blood cell formation; at other times the white cells alone are affected and a marked reduction in polymorphonuclear cells takes place, or even the thrombocytoplastic activity may be disturbed. Different names have been given to these conditions, for instance, aplastic anemia, agranulocytosis and thrombopenia, according to which element was involved. A combination in which the formations of white blood cells and platelets are affected has been referred to as aleukia haemorrhagica. There have been cases in which all of the various hemopoietic activities of the bone marrow were involved; such a disorder has been designated panmyelotoxicosis.

Certain drugs and chemical substances have been known to produce these effects. Among these are arsenic, arsphenamine, bismuth, aminopyrines and other benzene ring derivatives. Moreover, similar paralytic effects may be produced by various forms of radiation, such as roentgen rays, radium and thorium either after therapy or when poisoning has occurred from the entrance of large quantities of radiation into the body. In cases of certain severe septic conditions, such as diphtheria, typhoid and pneumonia, a toxic effect on the myeloid tissues with inhibition of activity may result. Moreover, there are a considerable number of diseases in which the changes in the bone marrow appear to be secondary to the main disease process.

When sulfanilamide was introduced and it was appreciated that it was a benzene ring derivative, it was naturally suspected that something in the way of a paralytic effect on the bone marrow would be produced. Exactly what form this would take could not be positively anticipated, though it was well known that benzene produced platelet deficiency and agranulocytosis. With the introduction of sulfanilamide and its derivatives and their rapid acceptance as common therapeutic agents, reports soon appeared in the literature of cases in which a marked reduction in the polymorphonuclear cells—that is, granulocytopenia—had occurred.

Peculiarly, this effect did not go hand in hand with the amount of the drug given. There have been cases reported in which death occurred and in which only 8 Gm. was administered. However, in a general way the quantity appeared to make a difference. In many cases of granulocytopenia a certain amount of aplastic anemia was observed; that is, the drug had apparently impaired not only white cell but also red blood cell formation. The question naturally arose: Would this drug also impede platelet formation with the production of a clinical picture simulating purpura haemorrhagica (Werlhof's disease)? Recently a case was reported by Russell and Page in which such a condition followed the administration of a comparatively small amount of the drug. Thus this new group of therapeutic agents under certain circumstances can bring about the whole series of so-called myelotoxic disturbances which have been recognized in the past. Moreover, it would appear that while the quantity of drug employed is in some cases a contributing factor, individual susceptibility is probably even more important.

Autopsy records at McGill University show 2 cases in which this marked disturbance of bone marrow activity has resulted from the use of these drugs. One was a case of gonorrhea, in which a profound granulocytopenia was produced. In the second case the drug was given in treatment of meningitis. On examination of the blood no indication of any paralytic effect on the bone marrow was found. The white blood cell count was 30,000. However, after use of the drug the count dropped to almost nothing and a severe anemia developed. Histologic examination of the bone marrow in cases of this condition usually reveals a rather characteristic picture. There is a deficiency in maturing myelocytes, while the immature root cells and syncytial reticuloendothelial elements are unusually conspicuous.

There is one other change in the blood which has been reported after administration of this drug, and that is a hemolytic form of anemia. This is a peculiar reaction to the drug which comes on very suddenly after a very small dosage and apparently in most cases in persons who have been treated for syphilis. In these persons there has occurred a rapid destruction of the red blood cells. They have a high white blood cell count and evidence of regeneration. The same effect is noted in the kidneys as in persons who have been given transfusions of incompatible blood.

I can give no explanation for the paralytic effect of these drugs on the bone marrow. However, I think there is a definite swing to the view that there is some sort of deficiency present. It may be that the drug combines in some way in the body with a substance necessary for hemopoiesis. This combination renders the necessary substance inactive, and the production of cells in the bone marrow suffers for want of it. Possibly the persons who show such marked changes in the blood after administration of an apparently insignificant quantity of the drug may have had a comparatively small available supply. I should not be surprised if it were found eventually that the effect of these drugs is not strictly a toxic paralysis but rather a deficiency paralysis due to removal of some important substance necessary for maturation of the proliferating elements of the bone marrow.

As far as treatment of persons with these toxic symptoms is concerned, from the hemopoietic standpoint it is of course advisable to stop administration of the drug. Transfusions may be employed. However, one should bear in mind that a number of cases have been reported in which transfusions in the presence of agranulocytosis seemed to be peculiarly incompatible. Pentnucleotide is to be recommended when the white blood cell count gets very low. Finally, in cases in which the hemolytic form of anemia is produced the urine should be kept alkaline to prevent the precipitation of acid hematin in the tubules.

Bacteriologic Aspects of Chemotherapy. DR. FRED SMITH.

While I was listening to Dr. Scriver's talk, it seemed to me that recent theorizing on the way the drugs work will be the new approach to the ideal drug. Colebrook suggested some months ago that the drugs operate on organisms

which are having a rather unhappy time in the body. The drugs do not really kill the organisms completely but rather interfere with their continued multiplication. That raises the point as to how the drugs interfere with the bacterial cell multiplication. Files realized the difficulty of the bacterial cell in multiplying, and he speculated on a method whereby amino acids might be synthesized into protein. There are amino acids which affect bacterial cell multiplication. With paraaminobenzoic acid in a mixture of blood and sulfanilamide the sulfanilamide has no effect. It is suggested that the bacterial cell uses a series of enzymes to synthesize amino acids into proteins and so to multiply: If a sulfanilamide compound chemically resembles a key amino acid, the corresponding enzyme may combine with the drug and so be blocked for combination with the amino acid.

Sulfapyridine is more or less a compound of sulfanilamide with nicotinic acid. Sulfathiazole similarly is derived from sulfanilamide and thiamine. In other words, these substances look extremely like essential metabolites in the lives of cells. Enzymes might be diverted to react with these drugs rather than with cell metabolites, and in this way bacteria might be prevented from synthesizing new cells. This suggests a line of investigation in the direction of making new drugs.

A second point is that thiamine and nicotinic acid are all important in the lives of tissue cells as well as bacterial cells. If one is going to introduce drugs resembling these important vitamins in the body in order to prevent such a substance as nicotinic acid from entering in the proper combinations for bacterial synthesis, one should not add nicotinic acid to the drug, for the fact is being relied on that the bacterial cells in the presence of sulfanilamide or one of its derivatives cannot use nicotinic acid. Moreover, tissue cells use the same nicotinic acid and other substances in their metabolsm, and sulfanilamide compounds eliminate this tissue metabolism too. This is an attempt to explain the mechanism of toxicity of these drugs both for the bacteria and for the sick person, and I suggest that the ways of use at the moment are perhaps not the best.

Laryngeal Stenosis in a Child Following Laryngeal Diphtheria. DR. D. H. BALLON.

A boy aged 18 months was admitted to the Royal Victoria Hospital on Oct. 9, 1939. He had laryngeal diphtheria in April 1939. A tracheotomy was done on May 2 because of his inability to retain the intubation tube. Dilations of the larynx under direct vision have been carried out repeatedly. Direct laryngoscopic examination on November 25 showed almost complete stenosis of the larynx. Subsequently, dilations up to a no. 28 bougie have been carried out. He now has a good glottic opening. The left cord moves freely, but the right cord is fixed. The tracheotomy tube is still in place.

Acute Laryngotracheitis Treated by Intubation, Tracheotomy and Sulfathiazole. DR. D. H. BALLON.

A boy aged 2½ years was admitted to the hospital on Dec. 26, 1940 with a history of a cold and respiratory distress for three days. He was intubated with a Dwyer tube at the Alexandra Hospital two hours before admission to the Royal Victoria Hospital.

On admission he had a temperature of 100 F., a pulse rate of 104 and a respiratory rate of 24. The child was transferred to this hospital because the dyspnea had not been satisfactorily relieved by intubation. On admission the child was pale and had an occasional croupy cough, but he was breathing well. The throat was mildly inflamed. The tonsils were enlarged. The chest was essentially normal except for harsh and prolonged breath sounds. There was an intubation tube in the larynx. Staphylococcus pyogenes was grown from the throat. The patient was transferred back to the Alexandra Hospital on December 28, where two days later he was extubated, but the tube had to be replaced because of

marked respiratory difficulty. Sulfathiazole, 7½ grains every six hours for four doses was given on December 31, and he received 97½ grains (6.3 Gm.) in the next five days.

He was readmitted to the Royal Victoria Hospital on Jan. 4, 1941, with findings as on the previous admission. The temperature was 100.3 F., the pulse rate 116 and the respiratory rate 36. A tracheotomy was performed on January 6, and the intubation tube was removed. Sulfathiazole therapy was continued. Considerable mucus was aspirated frequently through the tracheotomy tube. On January 24 laryngoscopic examination showed good movement of the vocal cords. The tracheotomy tube was removed that day and has not been replaced since. The child is free from respiratory distress and now talks normally.

A Patient Treated with Sulfanilamide for an Erysipeloid Lesion of the Face Showing Toxic Manifestations Assumed to be Due to the Use of the Drug. DR. KEITH O. HUTCHINSON.

A woman aged 31 was admitted to the Royal Victoria Hospital with a history of a cold in the head for one month and a painful cutaneous eruption about the left ear which had developed eight days prior to admission. She had been given small doses of sulfanilamide for the previous seven days, averaging 40 grains (2.6 Gm.) per day. Fever had been present for the past seven days, culminating with a rise in temperature to 106 F. on admission. Chills had been present for three days, headache for the past two days and nausea and vomiting for the past twenty-four hours with some drowsiness.

When she was admitted to the hospital the pharynx was mildly injected. The tonsils had been removed. The nose was clear, and no evidence of pus or mucopus was to be seen. There was no evidence of the erysipeloid rash about the left ear. There was a small gland in the left posterior cervical region that was palpable and tender. The general examination did not disclose anything of importance. The question of a diagnosis offered some difficulty. At first it was felt that the patient must have a septicemia. Blood taken for culture on admission subsequently proved to be normal. There was a concentration in the blood of 4.12 mg. of sulfanilamide per hundred cubic centimeters. There were 4,000,000 red blood cells, 15,400 white blood cells and 80 per cent hemoglobin, and the general blood picture conformed to that of an acute inflammatory process. There was no evidence of leukemia or other primary disease of the blood. A few pus cells were found in the urine but did not appear on repeated specimens. A roentgenogram of the chest was reported as normal, and blood agglutination tests for typhoid fever gave negative reactions.

In the first twelve hours after admission sulfanilamide to the extent of 120 grains (7.76 Gm.) was given in three doses. In view of all the normal findings and the fact that the temperature fell to normal in a four day period in a very striking manner after the cessation of sulfanilamide therapy, I came to the conclusion that toxic reaction to this drug was the correct diagnosis. The patient made an uninterrupted recovery and returned to work one month after discharge.

Acute Exacerbation of Chronic Suppurative Otitis Media with Thrombosis of the Lateral Sinus in a Child, Treated by Mastoidectomy, Ligation of the Internal Jugular Vein and Sulfapyridine. DR. KEITH O. HUTCHINSON.

A girl aged 10 years was admitted to the Royal Victoria Hospital on Nov. 26, 1940 with an acute exacerbation of a chronic suppurative otitis media on the right side. There had been discharge from this ear on and off for years, but the present exacerbation had been active for the past six months. She had nausea and vomiting for the past four days, pain in the right side of the neck and dizziness for twenty-four hours. The temperature on admission was 100 F., the pulse rate 112 and the respiratory rate 24. The child did not appear stuporous or acutely ill.

On examination of the right ear, there was some tenderness over the mastoid; foul-smelling discharge was seen coming from a small perforation in the postero-superior quadrant of the drum. The left ear was normal. The central nervous system showed hypoactivity of the deep reflexes, and the knee jerks were obtained with difficulty. The white blood cell count on admission was 12,000.

On the day following admission the child had a definite chill and appeared much worse. A lumbar puncture on this date showed an initial pressure of the spinal fluid of 300 mm. of water and a final pressure of 75 mm. Ten cubic centimeters of colorless fluid was withdrawn. A radical mastoid operation was carried out during the afternoon of November 27. The cortex was exposed and found intact. Entrance in the usual manner with hammer and chisel showed scarcely any pneumatic development, only a few tiny cells being present. The lateral sinus was quickly exposed and at once gave the impression that it was thrombosed, lying away from the bony walls. The antrum was very difficult to locate and finally was picked up by going through the posterior meatal wall. It contained definite cholesteatomatous debris. The lateral sinus was then incised and found to contain a blood clot. It was traced backward for about ¾ inch (1.9 cm.), when free bleeding occurred. As the child's condition was not good it was felt wiser to proceed at once with the ligation of the jugular vein.

The child made a good recovery from this infection, although two chills developed in the next forty-eight hours. The patient was given sulfapyridine. No positive bacteriologic findings were ever made. Subsequently bronchopneumonia developed in the right lung, and the roentgenograms gave the impression of multiple pulmonary abscesses. As this condition cleared up quickly, it would appear that this diagnosis was not sustained.

The second stage operation was carried out on Jan. 3, 1941, when it was planned to cut the usual plastic flap. When the granulating mastoid wound was opened the underlying lateral sinus began to bleed, and, while this was readily controlled by packing, it was impossible to elevate the wound flaps on account of the bleeding. Therefore, it was deemed wiser to attempt to make a small graft through the meatus, which was done. The child has been attending the clinic for observation, and there is still some discharge from the right ear.

A Patient with Acute Maxillary Sinusitis Showing Toxic Manifestations of Sulfanilamide Treatment. DR. W. J. MCNALLY.

A man aged 27 was admitted to the Royal Victoria Hospital on Dec. 5, 1940 with a mild infection in the right maxillary antrum, a small amount of nasal discharge and a temperature of 99 F. Cultures of material from the nose showed Staphylococcus pyogenes. The white blood cell count was 14,000.

On the day of admission sulfanilamide was prescribed, 90 grains (5.8 Gm.) per day. This dosage was continued for four days, at the end of which time the patient's temperature became elevated to 100 F. For the next three days the dose of sulfanilamide was reduced to 40 grains (2.6 Gm.) per day on account of a general feeling of nausea attributed to the drug. The nasal condition remained much the same. On December 12 a chill occurred in the morning and another in the afternoon. The first chill was slight; the second lasted for more than ten minutes. The temperature was elevated above 103 F. On the following day one chill occurred, lasting twenty-five minutes.

On December 14 sulfanilamide therapy was discontinued and the administration of sulfathiazole, 40 grains per day, started. The chills continued, and on December 15 the retinal veins were full and dark, the right retinal vein pulsating. The question of a thrombophlebitis was considered. It was also considered that these chills might be due to a drug reaction. The level of the drug in the blood was followed carefully and did not rise above 5.7 mg. per hundred cubic centimeters. The white blood cell count did not rise above 14,000. The urine was concentrated but otherwise normal throughout hospitalization. On December 15 all chemotherapy was discontinued, with rapid improvement thereafter. The patient was discharged on December 20 well.

Basal Cell and Squamous Cell Carcinoma of the Nose Treated by Roentgen Therapy. DR. W. J. MCNALLY.

A man aged 48 was admitted to the Royal Victoria Hospital on Jan. 4, 1941. He gave a history of having had a small hole in the bridge of his nose seven years previously and that four months before admission an extensive ulceration of the bridge of the nose had occurred.

On admission he had a large ulcerating area about 6 cm. from above downward and 4 cm. from side to side. This ulceration extended from the columella to within about 1 cm. of the glabella. The lower part of the ulcerating area consisted of a flap which had fallen down, completely covering the central portion of the upper lip. The left nostril was not obviously open, but a probe could be passed into the cavity corresponding to the left nostril. The right nostril opened into a mass measuring about 4 mm. in diameter. It was possible for him to breathe through this opening. The turbinates were not identified. A specimen for biopsy was taken on January 7 and was reported as mixed squamous and basal cell carcinoma. The Wassermann reaction of the blood was negative. Roentgen ray treatments were begun on Jan. 6, 1941.

Acute Suppurative Otitis Media in a Child with Impaired Myeloid Activity Following the Use of Sulfapyridine. DR. E. A. STUART.

A boy aged 8 years was admitted to the Royal Victoria Hospital on May 13, 1940 complaining of pain in the right ear, discharge from the same ear and slight elevation of temperature. Three weeks previously an acute suppurative otitis media on the right side had developed, with a temperature of 101 F., and he was treated with sulfanilamide on the assumption that the infection was streptococcic. After thirty-six hours the discomfort subsided, and within a week the ear was dry.

On admission to the hospital, culture of the discharge from the ear showed Pneumococcus type 1. On examination, the upper part of the right ear drum was inflamed and full. The hearing was considerably reduced. A small amount of mucopus was present in the ear canal. The tonsils and adenoid had been removed previously. The white blood cell count was 9,100. Sulfapyridine was given, 45 grains (2.9 Gm.) for one day, and the white blood cell count was reduced to 6,000. The dose was reduced to 22½ grains (1.45 Gm.) per day, and on the following day the white blood cell count was 4,400. Administration of the drug was discontinued, and 50,000 units of antipneumococcus serum (rabbit) was given in divided doses. Within two days the white blood cell count rose to 8,700, and 15 grains (1 Gm.) of sulfapyridine was given in divided doses over a period of twenty-four hours. This reduced the white blood cell count to 6,000, and no further drug was given. On May 27 the ear drum was intact and gray and definite improvement had occurred in the mastoid, as shown by roentgen ray examination. The hearing was practically normal. No further trouble was experienced.

Acute Mastoiditis with Acute Hemorrhagic Nephritis Possibly Due to the Use of Sulfanilamide. DR. E. A. STUART.

A girl aged 7 years was admitted to the Royal Victoria Hospital on Oct. 6, 1940, suffering from acute otitis media and mastoiditis, associated with stiffness of the neck, photophobia and hematuria. Three weeks previously the child had an acute infection in the left ear. The ear continued to discharge, although the initial elevation of temperature and general malaise lasted for only a few days. One week prior to admission the temperature rose to 103 F. and there was more discharge from the ear. Her physician prescribed sulfanilamide, which apparently was used for a couple of days, when the urine became dark. Administration of sulfanilamide was then discontinued, but the urine has become steadily darker. In the past week headaches have been marked, associated with photophobia. There has been no puffiness of the face and no edema of the legs.

On admission to the hospital the child appeared acutely ill, with a temperature of 103 F. She was lethargic. The left ear was discharging profusely. The superficial mastoid area was extremely tender. There were definite stiffness of the neck and photophobia, but no more positive signs of meningeal irritation were noted. The urine was bloody and on microscopic examination contained 1,000,000 red blood cells per cubic millimeter. The left mastoid was operated on shortly after the patient's admission, and the entire mastoid process was found to be broken down. The temperature remained elevated, between 102 F. and 104 F. for three days, after which it gradually subsided. The urine continued to show blood on gross examination for four days, after which it gradually cleared. Two weeks after admission red blood cells were rare in the urine. The white blood cell count on admission was 25,000. A lumbar puncture on October 9 showed 2,240 polymorphonuclear cells per cubic millimeter. A lumbar puncture was repeated on October 10, when the white blood cell count was 170, and on October 14, when it was reduced to 8. On October 8 the patient was given sulfathiazole, 30 grains (2 Gm.) per day for two days. Sulfapyridine, 30 grains per day, was administered for seven days. A blood transfusion was given on October 8. On November 5 the child was discharged from the hospital with the mastoid wound practically healed, the ear dry, the urine normal and the general condition good.

Opinions expressed by the pediatricians differed as to the nature of the renal disturbance. The history left a doubt as to whether sulfanilamide had been used before or after the onset of hematuria. If the hematuria was due to a hemorrhagic nephritis, it is interesting that it responded so well to treatment. If it was due to sulfanilamide, it is interesting to note the improvement when sulfathiazole and sulfapyridine were being administered. The infection was due to hemolytic streptococci.

A Case of Ulcerative Pharyngitis with Impaired Myeloid Activity Following the Use of Sulfanilamide for a Gonorrheal Infection. DR. G. E. TREMBLE.

A man aged 25 was admitted to the Royal Victoria Hospital on Nov. 12, 1940, complaining of a sore throat of ten days' duration with difficulty in swallowing and excessive salivation. On October 19 a gonorrheal infection was diagnosed, and for two weeks he was given sulfanilamide, about 300 grains (20 Gm.) being used.

On admission to the hospital the patient had a temperature of 103 F., a pulse rate of 128 and a respiratory rate of 28. He was pale and apathetic. His gums bled freely. He had a marked injection of the soft palate with pinpoint-sized ulcerations. He had a mild pharyngitis and a large hard lymph node in the left anterior triangle of the neck. His urine was normal. His white blood cell count was 1,600, of which 54 per cent were lymphocytes, 30 per cent monocytes, 14 per cent myelocytes and 2 per cent mature polymorphonucleocytes. The hemoglobin was 74 per cent. Culture of material from the throat showed Neisseria catarrhalis. The Wassermann reaction of the blood was negative. He was given pentnucleotide intramuscularly, and the ulcerations in the soft palate rapidly disappeared. On November 13 the white blood cell count was 4,250 and on November 15, 5,000; on November 18 a hemogram showed the white cell count to be 10,300 with 37 per cent polymorphonucleocytes.

It was the opinion of Dr. T. R. Waugh, of the department of pathology, that the changes in the blood picture were to be explained on the basis of a previously impaired myeloid activity which had undergone regenerative changes. The patient was discharged on November 19 cured.

Frontal and Ethmoid Sinusitis Treated by External Frontal Ethmoidectomy. DR. G. E. TREMBLE.

A man aged 33 was admitted to the Royal Victoria Hospital on Nov. 18, 1940, with a history of headache and pain in the left eye for eight days, swelling around

the left eye for five days and proptosis for two days with diplopia for two days. Since 1929, when he suffered a fracture of the mandible and the left frontal bone, he had had a left-sided facial tic.

On admission, he had a normal temperature; there was proptosis of the left eye to the extent of 1 cm.; the external ocular movements were limited in all directions; the eyelids were swollen and inflamed, and there was marked congestion of the nasal mucosa but no discharge. On November 19 a radical frontal ethmoidectomy was performed and a fistula was found in the lower medial angle of the left frontal sinus. Between this and the orbital contents there was a large amount of old blood and pus which extended to the back of the orbit. The anterior ethmoidal cells were removed. A large rubber tube was inserted into the frontal sinus through the nose, and the skin was sutured. This tube was removed in ten days, at which time the proptosis had disappeared, and two days later the patient was discharged.

Directory of Otolaryngologic Societies*

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City. Time: June 8-12, 1942.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 11-14, 1942.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.
Place: Hotel Dennis, Atlantic City, N. J. Time: June 8-9, 1942.

American Laryngological Association

President: Dr. T. E. Carmody, 277-16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: June 1-3, 1942.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.
Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.
Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.
Western—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr. E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

INDEX TO VOLUME 35

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Book Reviews and Society Transactions are indexed under these headings in their alphabetical order under the letters B and S, respectively.

Abnormalities and Deformities: See under names of diseases, organs and regions, as Ectodermal Defect; Esophagus; Harelip; Nose; etc.
Abscess: See also under names of organs and regions, as Brain, abscess; Epiglottis, abscess; Lungs, abscess; etc.
  peritonsillar, tonsillectomy in cases of, 335
Acid, experimental and clinical study of hemostasis by pectin; hemostatic action of methylic ester of polygalacturonic acid, 142
Adamantinoma of floor of right nostril; occurrence in child aged 5 years, *928
Adenoids, action of roentgen irradiations on pharyngeal tonsil, 505
  and tonsils, 504, *653
  giant lymph follicle hyperplasia of nasopharynx; review of literature; report of case, *572
  indications for removal of tonsils and adenoids in children, 135
  relation of tonsillectomy and adenoidectomy to incidence of poliomyelitis, 338
  role of tonsils and adenoids in scarlet fever, 342
  tonsil knife and adenotome for use with head in hyperextended position, *934
Adenoma of bronchus, *955
  primary pituitary adenoma and syndrome of cavernous sinus, 338
Agranulocytosis: See Granulocytopenia
Air, pressure; aerosinusitis, *107
  study of changes in temperature and water vapor content of respired air in nasal cavity, 820
Air Passages: See Respiratory Tract
Airplane: See Aviation and Aviators
Air Raids, prevention and treatment of acoustic trauma in war and in civil life, 172
Allen, K.: Giant lymph follicle hyperplasia of nasopharynx; review of literature; report of case, *572
Allergy: See Anaphylaxis and Allergy
American Medical Association, report of American Otological Society representative to committees on audiometers, hearing aids, acoustic measurements and terminology of American Medical Association and American Standards Association, 143
American Otological Society, report of representative to committees on audiometers, hearing aids, acoustic measurements and terminology of American Medical Association and American Standards Association, 143
American Standards Association, report of American Otological Society representative to committees on audiometers, hearing aids, acoustic measurements and terminology of American Medical Association and American Standards Association, 143
Amphetamine, treatment of blockage of eustachian tube by inflation with amphetamine vapor, *646
Amygdalectomy: See Tonsillectomy
Anaphylaxis and Allergy: See also Asthma; Hay Fever; etc.
  allergy as contributing factor to biologic deafness, *582
  experimental hypersensitivity of mucous membranes of upper respiratory tract, *918
Anaphylaxis and Allergy—Continued
  reagins in convalescent serum indicating its inhibiting effect, 140
  role of water balance in clinical manifestations of allergy, 672
Anesthesia, experiences with employment and dosage of scopolamine-dihydro-oxycodeinone hydrochloride (eukodal ephetonine) in practice of otorhinolaryngology, 674
  for tonsillectomy, *659
Angina: See Throat; Tonsils
  Agranulocytic: See Granulocytopenia
  Vincent's: See Fusospirochetosis
Anomalies: See under names of organs, diseases and regions, as Esophagus; Harelip; Nose; etc.
Antiferments, antienzymatic nature of sulfanilamide's bacteriostatic action, 672
Antrum: See Maxillary Sinus
Aphonia: See Voice
Apicitis: See Temporal Bone
Apparatus: See also Instruments
  hearing aid problems, 838
  study of changes in temperature and water vapor content of respired air in nasal cavity, 820
  treatment of blockage of eustachian tube by inflation with amphetamine vapor, *646
Aquaeductus Cochleae: See Temporal Bone
Arbuckle, M. F.: Modified combination of irradiation and surgical therapy in treatment of late cancer of throat, with reports of cases, 676
Arteries: See also Thrombosis; etc.
  carotid artery surgery, *30
Asthma: See also Anaphylaxis and Allergy
  bronchial, importance of chronic sinusitis in treatment of, 817
Atelectasis: See Lungs, collapse
Athletics, fatal ski stick injury of soft palate, 505
Atmosphere: See Air
Audiogram: See Hearing, tests
Audiometer: See Hearing, tests
Audition: See Hearing
Auditory Organ: See Ear
Aviation and Aviators, aerosinusitis, *107
  ear in flying, 174
Award, Casselberry, 133

Bacilli: See Bacteria
Bacteremia: See also Septicemia; Staphylococci; etc.
  Haemophilus influenzae type B in acute laryngitis with bacteremia, 336
Bacteria: See also Pneumococci; Staphylococci; Streptococci; etc.
  bacteriologic relationship of dental infection and chronic hyperplastic sinusitis, 985
  mucosus capsulatus group; septicemia due to Friedländer's bacillus; report of case following chronic otitis media complicated by sinus thrombosis; recovery following therapy with sulfanilamide and its derivatives, *772
Bacteriology; bacteriologic aspects of chemotherapy, 991
Ballon, D. H.: Acute laryngotracheitis treated by intubation, tracheotomy and sulfapyridine, 992
  Laryngeal stenosis in child following laryngeal diphtheria, 992
Basilar Membrane: See Labyrinth
Beatty, H. G.: Retractor for use in neck surgery, *651
Bell's Paralysis: See Paralysis, facial
Benzedrine: See Amphetamine
Berkove, A. B.: Adamantinoma of floor of right nostril; occurrence in child aged 5 years, *928

Bilharziasis: See Schistosomiasis
Bismuth and Bismuth Compounds, Therapy: See Tonsils
Black, W. C.: Chemotherapy of tuberculous otitis media, 158
Blahd, M.: Experimental hypersensitivity of mucous membranes of upper respiratory tract, *918
Blood coagulation, effect of nicotinic acid on, 508
Diseases: See Leukemia; etc.
distribution of sulfanilamide between blood and spinal fluid with reference to intraspinal treatment, 987
hematologic aspects of chemotherapy, 990
Boeck's Sarcoid: See Sarcoidosis
Bones, Conduction: See Hearing, tests
Diseases: See Osteomyelitis
marrow; acute suppurative otitis media in child with impaired myeloid activity following use of sulfapyridine, 995
marrow; ulcerative pharyngitis with impaired myeloid activity following use of sulfanilamide for gonorrheal infection, 996

Book Reviews:

Cancer of Face and Mouth; V. P. Blair, S. Moore and L. T. Byars, 511
Diseases of Ear, Nose and Throat: Principles and Practice of Otorhinolaryngology; F. L. Lederer, 353
Diseases of Nose, Throat and Ear: Handbook for Students and Practitioners; I. S. Hall, 843
Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions; W. P. Eagleton, 840
Nasal Sinuses: Anatomic and Clinical Consideration; O. E. Van Alyea, 841
Pathology of Oral Cavity; L. R. Cahn, 511
Surgical Treatment of Ethmofrontal Sinusitis by Means of Osteoplastic Method; R. A. Bergara, 842

Boros, E.: Cardiospasm; improved esophageal dilator, *799
Bougie, permanent bougie in treatment of acute corrosive esophagitis, 670
Brain: See also Meninges; etc.
abscess; traumatic perforation of right frontal sinus with pneumocephalus and brain abscess; report of case, 349
development of olfactory nerve and nervus terminalis in man, 828
Diseases: See Encephalitis
intracranial complications of tonsillar origin with reference to their incidence and pathogenesis, *530
tumors, diagnosis of, 341
Bronchi, adenoma of, *955
bronchography, *963
carcinoma of, *956
Foreign Bodies: See Foreign Bodies
lipoma of left main bronchus; report of case and review of literature, *868
obstruction, *943
present status in treatment of acute obstructive infections of larynx, trachea and bronchi, 669
results of treatment of tuberculosis of trachea and bronchi, 816
tracheobronchial tuberculosis, *945
Bronchiectasis, *949
pathogenesis of, 138
Bronchography: See under Bronchi
Bronner, C. E. C.: Presence and action of lysozyme in nasal mucus, 825
Brown, A. M.: Correction of facial defects with latex prostheses; technic, *720
Brunner, H.: Nasal glands, *183
Bryant, F. L.: Local use of sulfanilamide in otogenic infections, *281
Bunch, C. C.: Development of audiometer, 145
Burman, D.: Otorhinogenic meningitis; report of 58 cases, *687
Burman, H. J.: Otorhinogenic meningitis; report of 58 cases, *687
Calculus: See Salivary Glands
Caloric Test: See Labyrinth
Campbell, E. H.: Evaluation of labyrinth fenestration for chronic progressive deafness, 149
Campbell, P. A.: Aerosinusitis, *107
Cancer: See also under names of organs and regions, as Bronchi; Ear; Esophagus; Larynx; Maxillary Sinus; Nose; Throat; etc.
radiation damage to tissue and its repair, 987
Cardiospasm: See Stomach
Cardiovascular Diseases, dysphagia with disorders of heart and great vessels, 819
Cargile's Membrane: See Ear, tympanic membrane
Carmody, T. E.: Relation of otolaryngology to dental pathology, 683
Carotid Canal: See Temporal Bone
Cartilage, autogenous cartilage grafts; experimental study, 339
Casselberry Award, 133
Cavernous Sinus, primary pituitary adenoma and syndrome of, 338
Cerebrospinal Fever: See Meningitis
Cerebrospinal Fluid, distribution of sulfanilamide between blood and spinal fluid with reference to intraspinal treatment, 987
Chamberlain, D.: Occupational deafness; audiometric observations on aural fatigue and recovery; preliminary report, *595
Chancre of nasal septum, *932
Chemotherapy: See also under names of diseases, as Osteomyelitis; Otitis Media; Respiratory Tract, diseases; etc.
bacteriologic aspects of, 991
clinical aspects of, 988
hematologic aspects of, 990
Children, foreign bodies swallowed by, 141
injuries to nose; diagnosis and treatment, 684
recognition, treatment and prevention of hearing impairment in, 813
studies on pupils of Pennsylvania School for Deaf; mechanism of inheritance of deafness, *871
treatment and prevention of deafness in, 666
Choanae: See Nose
Chondroma of larynx, *640
transeptal removal of chondroma replacing sphenoid bone and sella, 831
Chordoma, *925
Cicatrix: See also under Esophagus
surgical management of postradiation scars and ulcers, 339
Ciocco, A.: Analysis of speech characteristics in deafened children, with observations on methods of training, 160
Studies on pupils of Pennsylvania School for Deaf; mechanism of inheritance of deafness, *871
Clerf, L. H.: Peroral endoscopy, *936
Coates, G. M.: Address of president of American Otological Association, 144
Cochlea: See Hearing; Labyrinth
Colds: See Respiratory Tract, diseases
Connell, E. S.: Local use of sulfanilamide and its derivatives in ear, nose and throat, *740
Cordray, D. P.: Hemangioma of temporal bone, 343
Corti's Organ: See Labyrinth
Cotton, receptacle for ready-made cotton wool ear wicks, *436
Courville, C. B.: Intracranial complications of tonsillar origin with reference to their incidence and pathogenesis, *530
Cracovaner, A. J.: Chancre of nasal septum, *932
Radical mastoidectomy with complete recovery in case of chronic lymphatic leukemia, *922
Cranium: See also Frontal Bone; Temporal Bone; etc.
intracranial complications of tonsillar origin with reference to their incidence and pathogenesis, *530

Cranium—Continued
neuroepithelioma in temporal bone; invasion of petrous and squamous portions, with extension into middle and posterior cranial fossae, *745
Crico-Arytenoid Joints: See under Larynx
Croup, Diphtheric: See Diphtheria, laryngeal
Cysts: See under names of organs and regions

Davis, A. H.: Foreign body (tooth) in pharynx, *434
Day, K. M.: Primary pseudocholesteatoma of ear, 679
Deaf-Mutism, organization of instruction in schools for deaf-mutes, 667
Deafness: See also Ear, diseases; Hearing; Otitis Media; Otosclerosis
analysis of speech characteristics in deafened children, with observations on methods of training, 160
anatomy and physiology of ear, *480
biologic, allergy as contributing factor to, *582
chronic progressive, including otosclerosis and diseases of inner ear, *291
conservation of hearing, *463
evaluation of labyrinth fenestration for chronic progressive deafness, 149
hearing aid problems, 838
hearing aids, *454
law of symmetric functional loss of hearing in inherited deafness, 504
occupational; audiometric observations on aural fatigue and recovery; preliminary report, *595
progressive amelioration of; president's address, 144
recognition, treatment and prevention of hearing impairment in children, 813
report of American Otological Society representative to committees on audiometers, hearing aids, acoustic measurements and terminology of American Medical Association and American Standards Association, 143
report of consultants on audiometers and hearing aids, 143
studies on pupils of Pennsylvania School for Deaf; mechanism of inheritance of deafness, *871
Tests: See Hearing, tests
therapy of nerve deafness and tinnitus aurium; use of large doses of thiamine hydrochloride and evaluation of results, with source of possible error in interpretation of improvement, *513
treatment and prevention in children, 666
use of radium in treatment by irradiation, *845
vitamin B_1 therapy for nerve deafness, 668
war deafness and its prevention, 332
Death, cochlear activity after death, 981
Deglutition, disorders; dysphagia with disorders of heart and great vessels, 819
Diabetes Mellitus, treatment and prognosis of aural infections complicated by severe diabetes, 680
Diphtheria, chronic tonsillitis in secondary syphilis; differential diagnosis from diphtheria and Vincent's infection; report of 23 cases, 134
laryngeal stenosis in child following laryngeal diphtheria, 992
postdiphtheritic stenosis of esophagus, 669
Directory of otolaryngologic societies, 182, 354, 512, 686, 844, 998
Distomiasis, Distoma hepaticum in nasal cavity, 818
Dowdy, A. H.: Use of radium in treatment of deafness by irradiation, *845
Dropsy, labyrinthine dropsy and Ménière's disease, *853
Duffner, G. J.: Chemotherapy of tuberculous otitis media, 158
Dworetzky, J. P.: Laryngeal tuberculosis; study of 500 cases of pulmonary tuberculosis, with résumé based on 28 years' experience, 682
Dwyer, J. G.: Newer conceptions of rhinootologic meningitis, 681
Dysphagia: See Deglutition, disorders

Eagle, W. W.: Sphenopalatine ganglion neuralgia, *66
Ear: See also Deafness; Hearing; Otorhinolaryngology; and under names of special structures, i. e., Labyrinth; Mastoid; etc.
anatomy and physiology of, *480
carcinoma of external auditory canal, 504
chronic suppurative otitis media on right, with labyrinthitis following removal of aural polyp, 344
Diseases: See also Meningitis, otitic; Otitis Media; Otosclerosis; etc.
diseases; local use of sulfanilamide in otogenic infections, *281
diseases; treatment and prognosis of aural infections complicated by severe diabetes, 680
Fistula: See Fistula
in experimental vitamin A deficiency, 981
in flying, 174
influence of nasopharyngeal hyperplasia on ear; histologic examination of hyperplastic lymph follicles after irradiation, 148
Internal: See Labyrinth
management of injuries to middle and internal ear, including fractures of temporal bone, 170
Middle: See Otitis Media; Vertigo, aural
newer conceptions of rhinootologic meningitis, 681
otosclerosis; symptoms in external and middle ear, 982
prevention and treatment of acoustic trauma in war and in civil life, 172
primary pseudocholesteatoma of, 679
receptacle for ready-made cotton wool ear wicks, *436
reconstruction of, 667
tuberculosis of, *221
tympanic membrane; closure of perforations of membrana tympani with Cargile's membrane, 980
Ectodermal Defect, nasal deformity in anhidrotic ectodermal dysplasia, *210
Electroaudiography: See Hearing, tests
Electrocoagulation: See under names of diseases, organs and regions, as Tonsils; etc.
Elwood, B. J.: Importance of control of pulmonary lesion when tuberculous tracheobronchitis coexists, *408
Emerson, E. B., Jr.: Use of radium in treatment of deafness by irradiation, *845
Encephalitis, localized nonsuppurative encephalitis secondary to infections of temporal bone and paranasal sinuses, with report of 4 cases, 140
Endocrine Glands, endocrine basis for otosclerosis, 681
Endoscopy, peroral, *936
Endothelioma of larynx, 670
Enzymes: See Antiferments
Epiglottis, abscess of, 136
early glossoepiglottic changes in congenital syphilis, 136
Epithelioma, tumors of larynx other than squamous cell epithelioma, 675
Equilibrium: See Labyrinth
Erysipelas of oral mucosa following tonsillectomy, 668
Erysipeloid, patient treated with sulfanilamide for erysipeloid lesion of face showing toxic manifestations assumed to be due to use of drug, 993
Esophagus: See also Deglutition
abnormalities; congenital malformations, *964
cancer of, *970
cardiospasm; improved esophageal dilator, *799
cicatricial stenosis, *965
fibrosis; cardiospasm, *967
fresh foreign body lesion combined with old foreign body healed into thyroid; misleading diagnosis; causative connection, 668
leiomyosarcoma of, *969

Esophagus—Continued
permanent bougie in treatment of acute corrosive esophagitis, 670
physiologic studies, *963
postdiphtheritic stenosis of, 669
stenosis, *965
syphilis of, *966
varix of, *966
Estrogens; estrogenic implants in treatment of atrophic rhinitis, *883
Ethmoid Bone, tumor of, 347
Ethmoid Sinus: See also Sinuses, Nasal
frontal and ethmoid sinusitis treated by external frontal ethmoidectomy, 996
Eukodal: See Anesthesia
Eustachian Tube, eustachian orifice; relation to landmarks in nose and nasopharynx, *649
treatment of blockage by inflation with amphetamine vapor, *646
Use of radium in treatment of deafness by irradiation, *845
Evans, M. G.: Hemangioma of temporal bone, 343
Exophthalmos: See Goiter, exophthalmic
Eyes, diseases; value of conservative management in rhinogenic orbital complications, 985
paralysis: primary pituitary adenoma and syndrome of cavernous sinus, 338
tonic deviations of eyes produced by movements of head, with reference to otolith reflexes; clinical observations, 138

Face, correction of facial defects with latex prostheses; technic, *720
fractures; maxillofacial injuries; important considerations, 673
Paralysis: See Paralysis, facial
reconstruction of facial defects with latex compound; results recently obtained, 987
Farrior, J. B.: Nasopharyngeal radium applicator, *811
Fatigue, Auditory: See Hearing
Fibroma, benign encapsulated, of larynx, *288
Fistula, congenital aural fistula, *431
evaluation of labyrinth fenestration for chronic progressive deafness, 149
Foreign Bodies; foreign body (tooth) in pharynx, *434
fresh foreign body lesion of esophagus combined with old foreign body healed into thyroid; misleading diagnosis; causative connection, 668
in air and food passages, *972
swallowed by children, 141
Fowler, E. P.: Hearing standards for acceptance, disability and discharge in military service (and in industry), 167
Report of American Otological Society representative to committees on audiometers, hearing aids, acoustic measurements and terminology of American Medical Association and American Standards Association, 143
Report of consultants on audiometers and hearing aids, 143
Fox, C. C.: Extensive carcinoma of maxillary sinus, 351
Fox, M. S.: Congenital aural fistula, *431
Fractures: See Jaws; Temporal Bone; etc.
Frank, I.: Experimental hypersensitivity of mucous membranes of upper respiratory tract, *918
Freeman, M. S.: Septicemia due to Friedländer's bacillus; report of case following chronic otitis media complicated by sinus thrombosis; recovery following therapy with sulfanilamide and its derivatives, *772
Frontal Bone, gunshot wound of frontal and temporal bones, 831
Frontal Sinus: See also Sinuses, Nasal
frontal and ethmoid sinusitis treated by external frontal ethmoidectomy, 996
indication and dangers of intranasal exposure of, 671
osteoma of, 671, 985
osteoma; report of case, with operation, 508
traumatic perforation of right frontal sinus with pneumocephalus and brain abscess; report of case, 349
Fusospirochetosis, chronic tonsillitis in secondary syphilis; differential diagnosis from diphtheria and Vincent's infection; report of 23 cases, 134
fusospirochetal organisms and tonsillitis; bismuth therapy of tonsillitis, *587
management of Plaut-Vincent's tonsil ulcer, 135

Galloway, T. C.: Transeptal removal of chondroma replacing sphenoid bone and sella, 831
Galvanic Test: See Labyrinth
Ganglion: See also Meckel's Ganglion
development of olfactory nerve and nervus terminalis in man, 828
Sphenopalatine: See Meckel's Ganglion
Gastroscopy: See Stomach
Glossitis: See under Tongue
Goiter, exophthalmic; vestibular examination of patients with exophthalmic goiter, 503
Gonorrhea, ulcerative pharyngitis with impaired myeloid activity following use of sulfanilamide for gonorrheal infection, 996
Gough, E. J.: Type III pneumococcic meningitis of otitic origin; recovery following operation and treatment with sulfathiazole and specific immune serum, *803
Gradenigo Syndrome: See Otitis Media
Graebner, H.: Chemotherapy in acute nondiphtheritic laryngotracheobronchitis, *523
Grafts: See Cartilage; Skin, grafts; etc.
Granulocytopenia, agranulocytosis associated with certain changes in nasal mucous membrane; report of case, 510
Grip: See Influenza
Grove, W. E.: Management of injuries to middle and internal ear, including fractures of temporal bone, 170
Growth, persistence of function of skin grafts through long periods of growth, 340
Guerry, Du P., III: Local use of sulfathiazole in management of simple mastoidectomy wounds, *399
Gunshot Wounds: See Wounds

Hallaron, J.: Giant lymph follicle hyperplasia of nasopharynx; review of literature; report of case, *572
Hammond, A. E.: Diagnosis and treatment of cancer of larynx with statistical review of 15 cases, *1
Hara, H. J.: Intracranial complications of tonsillar origin with reference to their incidence and pathogenesis, *530
Harelip, secondary repair of cleft lips and their nasal deformities, 815
Havens, F. Z.: Tumors of larynx other than squamous cell epithelioma, 675
Hay Fever: See also Anaphylaxis and Allergy
oral vs. parenteral pollen therapy; clinical study, 509
use of potassium chloride in treatment of allergic conditions, 140
Head: See also Cranium
tonic deviations of eyes produced by movements of head, with reference to otolith reflexes; clinical observations, 138
tonsil knife and adenotome for use with head in hyperextended position, *934
Hearing: See also Deafness
anatomy and physiology of ear, *480
and war, *471
Conservation: See under Deafness
functional examination of, *437
further objections to accepted interpretations of cochlear mechanics, *613
malingering and neuro-otologic considerations in combat, 173
occupational deafness; audiometric observations on aural fatigue and recovery; preliminary report, *595
reactions in ear to sound, 982
report of American Otological Society representative to committees on audiometers, hearing aids, acoustic measurements and terminology of American Medical Association and American Standards Association, 143

Hearing—Continued
report of consultants on audiometers and hearing aids, 143
standards for acceptance, disability and discharge in military service (and in industry), 167
tests, *437
tests; acoustic trauma in war and in peace, 834
tests; acute surgical mastoiditis; review of cases, with reference to postoperative hearing and to role of sulfanilamide and its derivatives in therapy, *250
tests; audiometers, *449
tests; clinical application of vestibular tests, *766
tests; development of audiometer, 145
tests; electroaudiography; analysis and interpretation of audiogram, *899
tests; increased sensitivity to bone-conducted sounds, *418
Heatly, C. A.: Use of radium in treatment of deafness by irradiation, *845
Heine-Medin's Disease: See Poliomyelitis
Hemangioma of temporal bone, 343
Hematology: See Hemopoietic System
Hematoma, subcutaneous, of neck following vocal strain, 506
Hemopoietic System, hematologic aspects of chemotherapy, 990
Hemoptysis, *945
Hemorrhage: See under Nephritis; Tonsillectomy; etc.
Hemostasis, experimental and clinical study of hemostasis by pectin; hemostatic action of methylic ester of polygalacturonic acid, 142
Hempstead, B. E.: Treatment and prognosis of aural infections complicated by severe diabetes, 680
Henner, R.: Endaural mastoidectomy; experience in series of 76 cases, *777
Gunshot wound of frontal and temporal bones, 831
Papilloma of tonsil and uvula, *810
Hernia: See under Larynx
Herpes zoster and Bell's palsy, 340
Hildebrand, A. G.: Treatment and prognosis of aural infections complicated by severe diabetes, 680
Hodgkin's Disease, *962
Holinger, P. H.: Chondroma of larynx, *640
Holmes, E. M.: Anatomy and physiology of ear, *480
Howell, K. M.: Experimental hypersensitivity of mucous membranes of upper respiratory tract, *918
Hubby, L. M.: Present conception of Gradenigo syndrome, 154
Hughson, W.: Analysis of speech characteristics in deafened children, with observations on methods of training, 160
Hearing aid problems, 838
Studies on pupils of Pennsylvania School for Deaf; mechanism of inheritance of deafness, *871
Hutchinson, K. O.: Acute suppurative otitis media with thrombosis of lateral sinus in child, treated by mastoidectomy, ligation of internal jugular vein and sulfapyridine, 993
Patient treated with sulfanilamide for erysipeloid lesion of face showing toxic manifestations assumed to be due to use of drug, 993
Hydrogen peroxide; local use of sulfanilamide powder and hydrogen peroxide in wound infections, 986
Hypophysis: See Pituitary Body

Imperatori, C. J.: Leonardo da Vinci's contribution to laryngology, rhinology and phonetics, 836
Industrial Diseases, occupational deafness; audiometric observations on aural fatigue and recovery; preliminary report, *595
Industry, hearing standards for acceptance, disability and discharge in military service (and in industry), 167
prevention and treatment of acoustic trauma in war and in civil life, 172
Infantile Paralysis: See Poliomyelitis
Infants, New-Born: See New-Born Infants
Infection: See also Bacteremia; Septicemia; etc.; and under names of bacteria, as Streptococci; etc.
treatment of follicular focal infection from standpoint of internist, with reference to electrocoagulation of tonsils, 336
Influenza, grippal tracheobronchitis and its treatment, 337
Haemophilus influenzae type B in acute laryngitis with bacteremia, 336
Injuries: See under names of organs and regions, as Ear; Face; Larynx; Nose; etc.
Instruments: See also Apparatus
nasopharyngeal radium applicator, *811
receptacle for ready-made cotton wool ear wicks, *436
retractor for use in neck surgery, *651
sphenoid sinus drill, *132
tonsil knife and adenotome for use with head in hyperextended position, *934
use of radium in treatment of deafness by irradiation, *845
Internal Secretions: See Endocrine Glands
Irrigation: See Sphenoid Sinus
Isaacs, H. J.: Congenital atresia of posterior nares; report of 2 cases, *603

Jaws, fractures; maxillofacial injuries; important considerations, 673
osteomyelitis of superior maxilla; report of case with recovery following chemotherapy, 832
use of temporary inexpensive bite block to determine relationship of closed bite and symptoms referable to temporomandibular joint, 156
Jennes, M. L.: Therapy of nerve deafness and tinnitus aurium; use of large doses of thiamine hydrochloride and evaluation of results, with source of possible error in interpretation of improvement, *513
Jones, A. C.: Carcinoma of larynx in girl aged 15, *426, 677
Jones, M. F.: Otosclerosis, with unusual pathologic findings, 165

Kaplan, I. I.: Malignant melanoma of nose, *85
Kepler, E. J.: Treatment and prognosis of aural infections complicated by severe diabetes, 680
Kerwin, R. W.: Carotid artery surgery, *30
Kimata, H.: Giant lymph follicle hyperplasia of nasopharynx; review of literature; report of case, *572
King, T.: Receptacle for ready-made cotton wool ear wicks, *436
Kopetzky, S. J.: Purulent otitis media, mastoiditis, sinus thrombosis and suppuration of petrous pyramid, *115

Labia Vocalia: See Vocal Cords
Labyrinth: See also Vertigo, aural
case for diagnosis; (1) labyrinthitis? (2) Ménière's syndrome? (3) subdural abscess? 347
chronic progressive deafness, including otosclerosis and diseases of inner ear, *291
chronic suppurative otitis media on right, with labyrinthitis following removal of aural polyp, 344
cochlear activity after death, 981
diagnosis of cerebral tumors, 341
evaluation of labyrinth fenestration for chronic progressive deafness, 149
Fistula: See Fistula
further objections to accepted interpretations of cochlear mechanics, *613
labyrinthine dropsy and Ménière's disease, *853
labyrinthitis from lightning, 333
management of injuries to middle and internal ear, including fractures of temporal bone, 170
melanosis of internal ear, *267

Labyrinth—Continued
nomenclature of vertical semicircular canals and resultant errors, 503
vestibular examination of patients with exophthalmic goiter, 503
Labyrinthitis: See under Labyrinth
Lacrimal Organs, transseptal operation on tear sac, 509
Laryngectomy: See under Larynx
Laryngocele: See Larynx, hernia
Laryngology: See also Otorhinolaryngology
Leonardo da Vinci's contribution to laryngology, rhinology and phonetics, 836
Laryngotracheobronchitis: See Respiratory Tract, diseases
Larynx: See also Vocal Cords
acute laryngotracheitis treated by intubation, tracheotomy and sulfapyridine, 992
anteroposterior roentgenograms of, 505
arrested development following irradiation for recurring papillomas, 822
benign encapsulated fibroma of, *288
bilharziasis of, *286
cancer; diagnosis and treatment with statistical review of 15 cases, *1
carcinoma in girl aged 15, *426, 677
carcinoma in young persons; report of 4 cases, 817
carcinoma; review of 170 consecutive cases, 816
chondroma of, *640
Diphtheria: See Diphtheria, laryngeal
endothelioma of, 670
Haemophilus influenzae type B in acute laryngitis with bacteremia, 336
hernia; external laryngocele, with operation, 506
hernia; infected laryngocele, 338
hypodynamia laryngis, *893
injuries of cricoid cartilage, 136
interrelations, diagnosis and treatment of benign and malignant tumors of larynx, with reference to prevention and cure of laryngeal cancer, 677
management of clinical problems involving larynx in infancy and childhood, 983
new artificial larynx, 669
paralysis of recurrent laryngeal nerve in human beings, 336
paralysis, prognosis following thyroidectomy, *732
pathogenesis of congenital inspiratory stridor, 506
present status in treatment of acute obstructive infections of larynx, trachea and bronchi, 669
stenosis in child following laryngeal diphtheria, 992
total laryngectomy, 984
tuberculosis; study of 500 cases of pulmonary tuberculosis, with résumé based on 28 years' experience, 682
tumors other than squamous cell epithelioma, 675
Lateral Sinus, Thrombosis: See Thrombosis
Latex, correction of facial defects with latex prostheses; technic, *720
reconstruction of facial defects with latex compound; results recently obtained, 987
Lawlor, E. F.: Tumor of ethmoid, 347
Lawrence, P. S.: Analysis of speech characteristics in deafened children, with observations on methods of training, 160
Leasure, J. K.: Clinical application of vestibular tests, *766
Lederer, F. L.: Melanosis of internal ear, *267
Leiomyosarcoma of esophagus, *969
Leonardo da Vinci: See da Vinci, Leonardo
Leshin, N.: Functional examination of hearing, *437
Leukemia, radical mastoidectomy with complete recovery in case of chronic lymphatic leukemia, *922
Lewin, M. L.: Nasal deformity in anhidrotic ectodermal dysplasia, *210
Lewis, E. R.: Malingering and neuro-otologic considerations in combat, 173
Lewis, J. F.: Fusospirochetal organisms and tonsillitis; bismuth therapy of tonsillitis, *587
Lewy, A.: Functional examination of hearing, *437
Osteomyelitis of superior maxilla; report of case with recovery following chemotherapy, 832
Lightning, labyrinthitis from, 333
Lindsay, J. R.: Labyrinthine dropsy and Ménière's disease, *853
Tuberculosis of ear, *221
Lipoma of left main bronchus; report of case and review of literature, *868
Lips: See Harelip
Lloyd, M. S.: Hypodynamia laryngis, *893
Lungs: See also Bronchi; Respiration; Respiratory Tract; etc.
abscess, *948
collapse; postoperative pulmonary atelectasis, *953
importance of control of pulmonary lesion when tuberculous tracheobronchitis coexists, *408
Tuberculosis: See Tuberculosis, pulmonary
Lymph Nodes, giant lymph follicle hyperplasia of nasopharynx; review of literature; report of case, *572
Lymphogranuloma, Hodgkin's: See Hodgkin's Disease
Lymphoid Tissue: See also Adenoids; Pharynx; Tonsils; etc.
influence of nasopharyngeal hyperplasia on ear; histologic examination of hyperplastic lymph follicles after irradiation, 148
Lymphosarcoma, primary, of tonsil, 815
Lysozyme, presence and action in nasal mucus, 825

McCall, J. W.: Septicemia due to Friedländer's bacillus; report of case following chronic otitis media complicated by sinus thrombosis; recovery following therapy with sulfanilamide and its derivatives, *772
McCall, R. E.: Case for diagnosis; (1) labyrinthitis?; (2) Ménière's syndrome? (3) subdural abscess? 347
Chronic suppurative otitis media on right side; acute otitis media with mastoiditis on left side, 346
Macfarlan, D.: Prevention and treatment of acoustic trauma in war and in civil life, 172
McNally, W. J.: Basal cell and squamous cell carcinoma of nose treated by roentgen therapy, 995
Patient with acute maxillary sinusitis showing toxic manifestations of sulfanilamide treatment, 994
Magnesium Sulfate: See Tinnitus Aurium
Malingering and neuro-otologic considerations in combat, 173
Malocclusion: See under Teeth
Mandible: See Jaws
Mangiaracina, A.: Eustachian orifice; relation to landmarks in nose and nasopharynx, *649
Mao, C.-Y.: Allergy as contributing factor to biologic deafness, *582
Marden, P. A.: Traumatic perforation of right frontal sinus with pneumocephalus and brain abscess; report of case, 349
Marks, R. F.: Sulfathiazole in treatment of sinusitis, *794
Martin, R. C.: Atypical facial neuralgia, 684, *735
Mastoid, Inflammation: See Mastoiditis
surgery; acute suppurative otitis media with thrombosis of lateral sinus in child, treated by mastoidectomy, ligation of internal jugular vein and sulfapyridine, 993
surgery; endaural mastoidectomy; experience in series of 76 cases, *777
surgery; gustatory sensibility of anterior two thirds of tongue in patients who have undergone radical mastoidectomy, 502
surgery; local use of sulfathiazole in management of simple mastoidectomy wounds, *399

Mastoid—Continued
surgery; new plastic flap for use in endaural radical mastoidectomy, 824
surgery; radical mastoidectomy with complete recovery in case of chronic lymphatic leukemia, *922
Mastoidectomy: See Mastoid, surgery
Mastoiditis: See also Otitis Media
acute surgical mastoiditis; review of cases, with reference to postoperative hearing and to role of sulfanilamide and its derivatives in therapy, *250
acute, with acute hemorrhagic nephritis possibly due to use of sulfanilamide, 995
and otitis media in children; bacteriologic studies with reference to pathogenicity of staphylococci, *631
artificial sinus thrombosis in disease of ear, 134
chronic suppurative otitis media on right side; acute otitis media with mastoiditis on left side, 346
diagnosis in infant by puncture of antrum, 502
local use of sulfanilamide in otogenic infections, *281
purulent otitis media, sinus thrombosis and suppuration of petrous pyramid, *115
radical mastoidectomy with complete recovery in case of chronic lymphatic leukemia, *922
treatment and prognosis of aural infections complicated by severe diabetes, 680
Matzkin, L. L.: Chondroma of larynx, *640
Maxillary Bone: See Jaws
Maxillary Sinus: See also Sinuses, Nasal
diagnosis and therapy of inflammation of, 507
diagnosis of mastoiditis in infant by puncture of antrum, 502
extensive carcinoma of, 351
patient with acute maxillary sinusitis showing toxic manifestations of sulfanilamide treatment, 994
sulfathiazole in treatment of sinusitis, *794
Meckel's Ganglion, sphenopalatine ganglion neuralgia, *66
Medicine, Military: See also Aviation and Aviators; Recruits; Wounds; etc.
hearing standards for acceptance, disability and discharge in military service (and in industry), 167
symposium on military subjects, 166
war deafness and its prevention, 332
what was wrong with otologic service in last war? 166
Medin-Heine's Disease: See Poliomyelitis
Melanin, melanosis of internal ear, *267
Melanoma, malignant, of nose, *85
Melanosis: See Melanin
Meltzer, P. E.: Hemangioma of temporal bone, 343
Membrane, Tympanic: See Ear, tympanic membrane
Ménière's Disease: See Vertigo, aural
Meninges, case for diagnosis: (1) labyrinthitis?; (2) Ménière's syndrome? (3) subdural abscess? 347
Meningitis, aseptic, resembling purulent meningitis and facial paralysis following acute suppurative otitis media, 502
otitic bacterial meningitis, *53
otitic; factors which may defeat treatment of otitic meningitis with sulfanilamide and its derivatives, 334
otorhinogenic; report of 58 cases, *687
petrositis with unusual complication, 814
rhinootologic, newer conceptions of, 681
type III pneumococcic meningitis of otitic origin; recovery following operation and treatment with sulfathiazole and specific immune serum, *803
use of sulfapyridine in Streptococcus viridans meningitis, 673
Meningococci: See under Meningitis
Menstruation, influence of menstrual cycle on ear, nose and throat diseases, 341
postoperative tonsillar hemorrhage and its relation to menstrual cycle, 136
Meteorology, seasonal variations influencing otitis media, 334
Michels, M. W.: Acute laryngotracheobronchitis in newborn infant; report of case with recovery following tracheotomy, *423
Military Medicine: See Medicine, Military
Mineral Oil: See Petrolatum, liquid
Morrison, L. F.: Type III pneumococcic meningitis of otitic origin; recovery following operation and treatment with sulfathiazole and specific immune serum, *803
Mosher, H. P.: What was wrong with otologic service in last war? 166
Mouth, erysipelas of oral mucosa following tonsillectomy, 668
Mucosa: See under Mouth; Nose; etc.
Mucous Membranes, experimental hypersensitivity of mucous membranes of upper respiratory tract, *918
Mulligan, E. J.: Prognosis of laryngeal paralysis following thyroidectomy, *732

Nares: See Nose
Nasopharynx, anatomic study as pathway to infracarotid portion of petrous pyramid, 666
eustachian orifice; relation to landmarks in nose and nasopharynx, *649
giant lymph follicle hyperplasia of; review of literature; report of case, *572
influence of nasopharyngeal hyperplasia on ear; histologic examination of hyperplastic lymph follicles after irradiation, 148
nasal atresias; surgical critique, 670
nasopharyngeal radium applicator, *811
Neck, retractor for use in neck surgery, *651
subcutaneous hematoma of neck following vocal strain, 506
Neoplasms: See Cancer; and under names of organs and regions
Nephritis, acute mastoiditis with acute hemorrhagic nephritis possibly due to use of sulfanilamide, 995
Nerves: See also Neuralgia; Neuritis; Paralysis
olfactory; development of olfactory nerve and nervus terminalis in man, 828
Paralysis: See under Paralysis
Recurrent: See Larynx, paralysis
therapy of nerve deafness and tinnitus aurium; use of large doses of thiamine hydrochloride and evalution of results, with source of possible error in interpretation of improvement, *513
vitamin B_1 therapy for nerve deafness, 668
Nervous System: See Brain; Cerebellum; Nerves; Reflex; etc.
Neter, E.: Otitis media and mastoiditis in children; bacteriologic studies with reference to pathogenicity of staphylococci, *631
Neuburger, F.: Otitis media and mastoiditis in children; bacteriologic studies with reference to pathogenicity of staphylococci, *631
Neuralgia, atypical facial neuralgia, 684, *735
sphenopalatine ganglion neuralgia, *66
Neuritis, optic; analysis of ocular fundus changes in otitic sinus thrombosis, 813
Neuroepithelioma in temporal bone; invasion of petrous and squamous portions, with extension into middle and posterior cranial fossae, *745
Neurofibromatosis, Recklinghausen's disease in otology, 164
Newborn Infants, acute laryngotracheobronchitis in; report of case with recovery following tracheotomy, *423
hypodynamia laryngis, *893
Nicotinic Acid, effect on blood coagulation, 508
Noise, acoustic trauma in war and in peace, 834
occupational deafness; audiometric observations on aural fatigue and recovery; preliminary report, *595
prevention and treatment of acoustic trauma in war and in civil life, 172
war deafness and its prevention, 332

Nomenclature of vertical semicircular canals and resultant errors, 503
Nose: See also Nasopharynx; Otorhinolaryngology; Rhinitis; etc.
Accessory Sinuses: See Sinuses, Nasal
adamantinoma of floor of right nostril; occurrence in child aged 5 years, *928
agranulocytosis associated with certain changes in nasal mucous membrane; report of case, 510
cancer; basal cell and squamous cell carcinoma of nose treated by roentgen therapy, 995
chancre of nasal septum, *932
deformities; congenital atresia of posterior nares; report of 2 cases, *603
deformities; nasal atresias; surgical critique, 670
deformities; secondary repair of cleft lips and their nasal deformities, 815
deformities; sphenopalatine ganglion neuralgia (including role of nasal deformities), *66
deformity in anhidrotic ectodermal dysplasia, *210
eustachian orifice; relation to landmarks in nose and nasopharynx, *649
hard papilloma of, 508
indication and dangers of intranasal exposure of frontal sinus, 671
Inflammation: See Rhinitis
injuries in children; diagnosis and treatment, 684
malignant melanoma of, *85
nasal glands, *183
newer conceptions of rhinootologic meningitis, 681
presence and action of lysozyme in nasal mucus, 825
structure of vessels in nasal mucosa of respiratory region, 671
study of changes in temperature and water vapor content of respired air in nasal cavity, 820
surgery, corrective, of nasal tip, 670
surgery; postoperative angina, 505
tuberculous osteoperiostitis of nasal bone, 818
Nystagmus: See Labyrinth
Caloric: See Labyrinth

Occupational Diseases: See Industrial Diseases
Olfactory Bulb: See Nerves, olfactory
Ophthalmoplegia: See Eyes, paralysis
Osteoma of frontal sinus, 671, 985
of frontal sinus; report of case, with operation, 508
Osteomyelitis, massive, as complication of pansinusitis, 351
of superior maxilla; report of case with recovery following chemotherapy, 832
Osteoperiostitis, tuberculous, of nasal bone, 818
Otitis Media: See also Mastoiditis
acute suppurative otitis media in child with impaired myeloid activity following use of sulfapyridine, 995
acute suppurative otitis media with thrombosis of lateral sinus in child, treated by mastoidectomy, ligation of internal jugular vein and sulfapyridine, 993
and lobar pneumonia in young children, 134
and mastoiditis in children; bacteriologic studies with reference to pathogenicity of staphylococci, *631
aseptic meningitis resembling purulent meningitis and facial paralysis following acute suppurative otitis media, 502
chronic suppurative otitis media on right side; acute otitis media with mastoiditis on left side, 346
chronic suppurative otitis media on right, with labyrinthitis following removal of aural polyp, 344
diagnostic significance of partial paralysis of facial nerve in chronic suppurative otitis media, 980
factors which may defeat treatment of otitic meningitis with sulfanilamide and its derivatives, 334
Otitis Media—Continued
local use of sulfanilamide in otogenic infections, *281
otitic bacterial meningitis, *53
otorhinogenic meningitis; report of 58 cases, *687
petrositis with unusual complication, 814
present conception of Gradenigo syndrome, 154
purulent, mastoiditis, sinus thrombosis and suppuration of petrous pyramid, *115
relation between pneumatization of temporal bone and complications of acute middle ear infection, 333
seasonal variations influencing, 334
septicemia due to Friedländer's bacillus; report of case following chronic otitis media complicated by sinus thrombosis; recovery following therapy with sulfanilamide and its derivatives, *772
treatment and prognosis of aural infections complicated by severe diabetes, 680
tuberculosis of ear, *221
tuberculous, chemotherapy of, 158
type III pneumococcic meningitis of otitic origin; recovery following operation and treatment with sulfathiazole and specific immune serum, *803
Oto-Audion: See Hearing, tests
Otolaryngologic societies, directory of, 182, 354, 512, 686, 844, 998
Otolaryngology: See also Otorhinolaryngology
relation to dental pathology, 683
Otology: See also Otorhinolaryngology
Recklinghausen's disease in, 164
what was wrong with otologic service in last war? 166
Otorhinolaryngology: See also Otology
experiences with employment and dosage of scopolamine-dihydro-oxycodeinone hydrochloride (eukodal ephetonine) in practice of, 674
influence of menstrual cycle on ear, nose and throat diseases, 341
Leonardo da Vinci's contribution to laryngology, rhinology and phonetics, 836
local use of sulfanilamide and its derivatives in ear, nose and throat, *740
Otosclerosis, chronic progressive deafness, including otosclerosis and diseases of inner ear, *291
endocrine basis for, 681
symptoms in external and middle ear, 982
with unusual pathologic findings, 165
Outpatient Department, review of 3,000 operations on outpatients performed in office, 673
Ozena: See Rhinitis, atrophic

Pagliughi, J. J.: Importance of control of pulmonary lesion when tuberculous tracheobronchitis coexists, *408
Palate, fatal ski stick injury of soft palate, 505
Palmer, C. E.: Studies on pupils of Pennsylvania School for Deaf; mechanism of inheritance of deafness, *871
Pansinusitis: See Sinuses, Nasal
Papilloma, arrested development of larynx following irradiation for recurring papillomas, 822
hard, of nose, 508
of tonsil and uvula, *810
Paralysis: See also Eyes, paralysis; Larynx, paralysis; Poliomyelitis; etc.
abducens; petrositis with unusual complication, 814
abducens; present conception of Gradenigo syndrome, 154
facial; aseptic meningitis resembling purulent meningitis and facial paralysis following acute suppurative otitis media, 502
facial; Bell's palsy and herpes zoster, 340
facial; diagnostic significance of partial paralysis of facial nerve in chronic suppurative otitis media, 980
Infantile: See Poliomyelitis
Laryngeal: See Larynx, paralysis
Recurrent: See Larynx, paralysis
Parkhill, E. M.: Tumors of larynx other than squamous cell epithelioma, 675

Pearson, A. A.: Development of olfactory nerve and nervus terminalis in man, 828
Pectin, experimental and clinical study of hemostasis by pectin; hemostatic action of methylic ester of polygalacturonic acid, 142
Peele, J. C.: Giant lymph follicle hyperplasia of nasopharynx; review of literature; report of case, *572
Pembleton, W. E.: Lipoma of left main bronchus; report of case and review of literature, *868
Pennsylvania School for Deaf, studies on pupils of; mechanism of inheritance of deafness, *871
Peritonsillitis: See Abscess, peritonsillar
Perlman, H. B.: Acoustic trauma in war and in peace, 834
Petrolatum, liquid; utilization of vitamin A added to mineral oil, 818
Petrositis: See under Temporal Bone
Petrous Bone: See Temporal Bone
Pharynx: See also Nasopharynx
aphthous pharyngitis, 814
foreign body (tooth) in, *434
ulcerative pharyngitis with impaired myeloid activity following use of sulfanilamide for gonorrheal infection, 996
x-ray treatment in infections of tonsils and pharynx, 504
Phonation: See Voice
Phonetics: See under Voice
Pituitary Body, primary pituitary adenoma and syndrome of cavernous sinus, 338
Pituitary Preparations, endocrine basis for otosclerosis, 681
Plastic Surgery: See Surgery, plastic
Plaut-Vincent's Infection: See Fusospirochetosis
Pneumocephalus, traumatic perforation of right frontal sinus with pneumocephalus and brain abscess; report of case, 349
Pneumococci: See also under Meningitis
angina of pneumococcic origin, 335
type III pneumococcic meningitis of otitic origin; recovery following operation and treatment with sulfathiazole and specific immune serum, *803
Pneumonia, lobar, and otitis media in young children, 134
relation between acute pneumonopathy and paranasal sinuses, 507
Pohlman, A. G.: Further objections to accepted interpretations of cochlear mechanics, *613
Increased sensitivity to bone-conducted sounds, *418
Poirier, G. H.: Tumor of ethmoid, 347
Poisons and Poisoning: See under names of substances
Poliomyelitis, relation of tonsillectomy and adenoidectomy to incidence of, 338
relation of tonsillectomy to, *91
Pollinosis: See Hay Fever
Polyp: See under Ear
Poppen, J. R.: Ear in flying, 174
Potassium Chloride: See Hay Fever
Potter, B. P.: Importance of control of pulmonary lesion when tuberculous tracheobronchitis coexists, *408
Pregnancy, endothelioma of larynx, 670
Pressman, J. J.: Physiology of vocal cords in phonation and respiration, *355
Priest, R. E.: Acute surgical mastoiditis; review of cases, with reference to postoperative hearing and to role of sulfanilamide and its derivatives in therapy, *250
Proctor, B.: Tuberculosis of ear, *221
Pseudocholesteatoma, primary, of ear, 679
Purpura, thrombocytopenic purpura due to sulfapyridine, 819
Putney, F. J.: Local use of sulfathiazole in management of simple mastoidectomy wounds, *399
Peroral endoscopy, *936

Quevedo, J.: Chronic suppurative otitis media on right, with labyrinthitis following removal of aural polyp, 344

Radiations: See also under names of various organs and diseases
damage to tissue and its repair, 987
Radium: See also Radiations
arrested development of larynx following irradiation for recurring papillomas, 822
nasopharyngeal radium applicator, *811
Therapy: See Deafness
Reagins: See Anaphylaxis and Allergy
Recklinghausen's Disease: See Neurofibromatosis
Recruits, hearing and war, *471
Reflex, tonic deviations of eyes produced by movements of head, with special reference to otolith reflexes; clinical observations, 138
Reich, C.: Radical mastoidectomy with complete recovery in case of chronic lymphatic leukemia, *922
Respiration, hypodynamia laryngis, *893
physiology of vocal cords in phonation and respiration, *355
study of changes in temperature and water vapor content of respired air in nasal cavity, 820
Respiratory Tract: See also Bronchi; Nasopharynx; Nose; Pharynx; etc.
anatomic and physiologic considerations, *936
diseases; acute laryngotracheobronchitis, *938
diseases; acute laryngotracheobronchitis in newborn infant; report of case with recovery following tracheotomy, *423
diseases; chemotherapy in acute nondiphtheritic laryngotracheobronchitis, *523
diseases; intranasal vaccine spray; its use in prophylaxis against common cold, 817
diseases; sulfanilamide and sulfapyridine in treatment of acute laryngotracheobronchitis, 816
Distoma hepaticum in nasal cavity, 818
experimental hypersensitivity of mucous membranes of upper respiratory tract, *918
foreign bodies in air and food passages, *972
scleroma in Slovakia, 820
treatment of upper respiratory tract infections in children, 141
Retractor for use in neck surgery, *651
Rheumatism, therapeutic effect of tonsillectomy on, 668
Rhinitis: See also Respiratory Tract, diseases
atrophic, estrogenic implants in treatment of, *883
atrophic; surgical treatment for ozena, 137
Rhinology: See Otorhinolaryngology
Rhinoplasty: See Nose, surgery
Ribs, autogenous cartilage grafts; experimental study, 339
Richardson, G. A.: Nasopharyngeal radium applicator, *811
Richardson, J. R.: Anatomy and physiology of ear, *480
Risch, O. C.: Laryngeal tuberculosis; study of 500 cases of pulmonary tuberculosis, with résumé based on 28 years' experience, 682
Robb, J. M.: Endocrine basis for otosclerosis, 681
Roberts, W. S.: Chronic progressive deafness, including otosclerosis and diseases of inner ear, *291
Roentgen Rays, surgical management of postradiation scars and ulcers, 339
Therapy: See under various diseases and organs, as Nose, cancer; Pharynx; etc.
Rosenbluth, M.: Otorhinogenic meningitis; report of 58 cases, *687
Rosenvold, L. K.: Estrogenic implants in treatment of atrophic rhinitis, *883

Sacco, A. G.: Importance of control of pulmonary lesion when tuberculous tracheobronchitis coexists, *408
Salinger, S.: Arrested development of larynx following irradiation for recurring papillomas, 822
Injuries to nose in children; diagnosis and treatment, 684
Salivary Glands, calculus of, 678, *761
Sarcoidosis, *963

Sarcoma: See Lymphosarcoma; and under names of organs and regions
Scala Tympani: See Labyrinth
Scarlet Fever: See also Streptococci
role of tonsils and adenoids in, 342
Schenck, H. P.: Influence of nasopharyngeal hyperplasia on ear; histologic examination of hyperplastic lymph follicles after irradiation, 148
Schistosomiasis, bilharziasis of larynx, *286
Schools, organization of instruction in schools for deaf-mutes, 667
Schwartz, A. A.: Congenital atresia of posterior nares; report of 2 cases, *603
Scleroma: See Respiratory Tract
Scriver, W.: Clinical aspects of chemotherapy, 988
Secretions, Internal: See Endocrine Glands
Sella Turcica, chordoma, *925
transeptal removal of chondroma replacing sphenoid bone and sella, 831
Semicircular Canal: See Labyrinth
Septicemia: See also Bacteremia; and under specific organisms, as Staphylococci; Streptococci; etc.
due to Friedländer's bacillus; report of case following chronic otitis media complicated by sinus thrombosis; recovery following therapy with sulfanilamide and its derivatives, *772
Septum, Nasal: See under Nose
Serum, reagins in convalescent serum indicating its inhibiting effect, 140
Therapy: See Otitis Media
Seydell, E. M.: Relation of tonsillectomy to poliomyelitis, *91
Shaheen, H. B.: Bilharziasis of larynx, *286
Shambaugh, G. E., Jr.: Chronic progressive deafness, including otosclerosis and diseases of inner ear, *291
New plastic flap for use in endaural radical mastoidectomy, 824
Therapy of nerve deafness and tinnitus aurium; use of large doses of thiamine hydrochloride and evaluation of results, with source of possible error in interpretation of improvement, *513
Tonsil knife and adenotome for use with head in hyperextended position, *934
Short, M. L.: Otitis media and mastoiditis in children; bacteriologic studies with reference to pathogenicity of staphylococci, *631
Short Wave Therapy: See Sinuses, Nasal
Singing, tonsillectomy and its effect on singing voice, *915
Singleton, J. D.: Tonsils and adenoids, *653
Sinus, Cavernous: See Cavernous Sinus
Ethmoid: See Ethmoid Sinus
Frontal: See Frontal Sinus
Maxillary: See Maxillary Sinus
Nasal: See Sinuses, Nasal
Sphenoid: See Sphenoid Sinus
Thrombosis: See under Thrombosis
Sinuses, Nasal: See also Ethmoid Sinus; Frontal Sinus; Maxillary Sinus; Sphenoid Sinus; etc.
aerosinusitis, *107
bacteriologic relationship of dental infection and chronic hyperplastic sinusitis, 985
chronic sinus infection in children, 137
complications incident to paranasal sinusitis, 506
importance of chronic sinusitis in treatment of bronchial asthma, 817
localized nonsuppurative encephalitis secondary to infections of temporal bone and paranasal sinuses, with report of 4 cases, 140
massive osteomyelitis as complication of pansinusitis, 351
relation between acute pneumonopathy and paranasal sinuses, 507
short wave therapy of acute purulent sinusitis, 508
sinusitis in children, 137
specific vaccine therapy in treatment of sinusitis, *623
Sinuses, Nasal—Continued
value of conservative management in rhinogenic orbital complications, 985
Skiing: See Athletics
Skillern, S. R., Jr.: Massive osteomyelitis as complication of pansinusitis, 351
Skin, grafts, persistence of function through long periods of growth, 340
new plastic flap for use in endaural radical mastoidectomy, 824
treatment of trauma of skin and subcutaneous tissues, 340
Skull: See Cranium
Smith, C. H.: Recklinghausen's disease in otology, 164
Smith, F.: Bacteriologic aspects of chemotherapy, 991
Smith, H. D.: Sphenoid sinus drill, *132
Societies, national, directory of, 182, 354, 512, 686, 844, 998
North Dakota Academy of Ophthalmology and Oto-Laryngology, 133
otolaryngologic, directory of, 182, 354, 512, 686, 844, 998
report of American Otological Society representative to committees on audiometers, hearing aids, acoustic measurements and terminology of American Medical Association and American Standards Association, 143

Society Transactions:

American Laryngological, Rhinological and Otological Society, Inc., 675
American Otological Society, Inc., 143
Chicago Laryngological and Otological Society, 822
College of Physicians of Philadelphia, Section on Otolaryngology, 349, 836
Massachusetts Eye and Ear Infirmary, 343
Montreal Medico-Chirurgical Society, Section of Otolaryngology, 988

Solis-Cohen, M.: Specific vaccine therapy in treatment of sinusitis, *623
Soper, G. R.: Transeptal removal of chondroma replacing sphenoid bone and sella, 831
Speech: See also Voice
analysis of speech characteristics in deafened children, with observations on methods of training, 160
Spencer, F. R.: Chemotherapy of tuberculous otitis media, 158
Sphenoid Bone, transeptal removal of chondroma replacing sphenoid bone and sella, 831
Sphenoid Sinus: See also Sinuses, Nasal
drill, *132
safe and simple method of irrigation, *755
Sphenopalatine Ganglion: See Meckel's Ganglion
Spinal Fluid: See Cerebrospinal Fluid
Sports: See Athletics
Squamitis: See under Temporal Bone
Staphylococci, otitis media and mastoiditis in children; bacteriologic studies with reference to pathogenicity of staphylococci, *631
Stomach, cardiospasm; improved esophageal dilator, *799
fibrosis of esophagus; cardiospasm, *967
gastroscopy, *977
Streptococci: See also Scarlet Fever
some aspects of infection; review, 141
use of sulfapyridine in Streptococcus viridans meningitis, 673
Stridor, Laryngeal: See under Larynx
Stuart, E. A.: Acute mastoiditis with acute hemorrhagic nephritis possibly due to use of sulfanilamide, 995
Acute suppurative otitis media in child with impaired myeloid activity following use of sulfapyridine, 995
Stutsman, A. C.: Modified combination of irradiation and surgical therapy in treatment of late cancer of throat, with reports of cases, 676
Sublingual Glands: See Salivary Glands

Submaxillary Gland, Calculi: See under Salivary Glands
Sulfanilamide and Sulfanilamide Derivatives, acute mastoiditis with acute hemorrhagic nephritis possibly due to use of sulfanilamide, 995
acute suppurative otitis media in child with impaired myeloid activity following use of sulfapyridine, 995
antienzymatic nature of sulfanilamide's bacteriostatic action, 672
bacteriologic aspects of chemotherapy, 991
clinical aspects of chemotherapy, 988
distribution of sulfanilamide between blood and spinal fluid with reference to intraspinal treatment, 987
hematologic aspects of chemotherapy, 990
local use of sulfanilamide and its derivatives in ear, nose and throat, *740
local use of sulfanilamide powder and hydrogen peroxide in wound infections, 986
patient treated with sulfanilamide for erysipeloid lesion of face showing toxic manifestations assumed to be due to use of drug, 993
patient with acute maxillary sinusitis showing toxic manifestations of sulfanilamide treatment, 994
Therapy: See Gonorrhea; Mastoiditis; Meningitis; Otitis Media; Scarlet Fever; Throat
thrombocytopenic purpura due to sulfapyridine, 819
ulcerative pharyngitis with impaired myeloid activity following use of sulfanilamide for gonorrheal infection, 996
Sulfapyridine, Therapy: See Meningitis; Thrombosis
Sulfathiazole, Therapy: See under Maxillary Sinus
Sulman, L. D.: Treatment of blockage of eustachian tube by inflation with amphetamine vapor, *646
Surgery, Plastic: See also under Face; Nose; etc.
plastic; new plastic flap for use in endaural radical mastoidectomy, 824
review of 3,000 operations on outpatients performed in office, 673
Swallowing: See Deglutition
Sweat Glands, nasal deformity in anhidrotic ectodermal dysplasia, *210
Symposium on military subjects, 166
Syphilis: See also under names of organs and regions, as Esophagus; etc.
chronic tonsillitis in secondary syphilis; differential diagnosis from diphtheria and Vincent's infection; report of 23 cases, 134
congenital, early glossoepiglottic changes in, 136

Taste, gustatory sensibility of anterior two thirds of tongue in patients who have undergone radical mastoidectomy, 502
Teeth, bacteriologic relationship of dental infection and chronic hyperplastic sinusitis, 985
foreign body (tooth) in pharynx, *434
relation of otolaryngology to dental pathology, 683
use of temporary inexpensive bite block to determine relationship of closed bite and symptoms referable to temporomandibular joint, 156
Temperature, study of changes in temperature and water vapor content of respired air in nasal cavity, 820
Temporal Bone, anatomic study of nasopharynx as pathway to infracarotid portion of petrous pyramid, 666
gunshot wound of frontal and temporal bones, 831
hemangioma of, 343
localized nonsuppurative encephalitis secondary to infections of temporal bone and paranasal sinuses, with report of 4 cases, 146
management of injuries to middle and internal ear, including fractures of, 170
Temporal Bone—Continued
neuroepithelioma in; invasion of petrous and squamous portions, with extension into middle and posterior cranial fossae, *745
petrositis with unusual complication, 814
primary and secondary tumors of, 334
purulent otitis media, mastoiditis, sinus thrombosis and suppuration of petrous pyramid, *115
relation between pneumatization of temporal bone and complications of acute middle ear infection, 333
tuberculosis of ear, *221
Temporomandibular Joint: See Jaws
Thiamine hydrochloride; therapy of nerve deafness and tinnitus aurium; use of large doses of thiamine hydrochloride and evaluation of results, with source of possible error in interpretation of improvement, *513
vitamin B_1 therapy for nerve deafness, 668
Tholen, E.: Calculus of salivary glands, 678, *761
Thorax: See Heart; Lungs; etc.
Throat: See also Larynx; Pharynx; Tonsils; etc.
angina of pneumococcic origin, 335
cancer; modified combination of irradiation and surgical therapy in treatment of late cancer of throat, with reports of cases, 676
Thrombosis, acute suppurative otitis media with thrombosis of lateral sinus in child, treated by mastoidectomy, ligation of internal jugular vein and sulfapyridine, 993
analysis of ocular fundus changes in otitic sinus thrombosis, 813
artificial sinus thrombosis in disease of ear, 134
purulent otitis media, mastoiditis, sinus thrombosis and suppuration of petrous pyramid, *115
septicemia due to Friedländer's bacillus; report of case following chronic otitis media complicated by sinus thrombosis; recovery following therapy with sulfanilamide and its derivatives, *772
Thyroid: See also Goiter
fresh foreign body lesion of esophagus combined with old foreign body healed into thyroid; misleading diagnosis; causative connection, 668
prognosis of laryngeal paralysis following thyroidectomy, *732
Tinnitus Aurium in light of recent research, 333
magnesium sulfate solution for treatment of, 814
therapy of nerve deafness and tinnitus aurium; use of large doses of thiamine hydrochloride and evaluation of results, with source of possible error in interpretation of improvement, *513
Tobey, H. G.: Case for diagnosis: (1) labyrinthitis?; (2) Ménière's syndrome? (3) subdural abscess? 347
Chronic suppurative otitis media on right side; acute otitis media with mastoiditis on left side, 346
Tongue, early glossoepiglottic changes in congenital syphilis, 136
Tonsillectomy and its effect on singing voice, *915
anesthesia for, *659
erysipelas of oral mucosa following tonsillectomy, 668
experiences with employment and dosage of scopolamine-dihydro-oxycodeinone hydrochloride (eukodal ephetonine) in practice of otorhinolaryngology, 674
in cases of peritonsillar abscess, 335
indications for removal of tonsils and adenoids in children, 135
postoperative tonsillar hemorrhage and its relation to menstrual cycle, 136
relation of tonsillectomy and adenoidectomy to incidence of poliomyelitis, 338
relation to poliomyelitis, *91
review of 3,000 operations on outpatients performed in office, 673
therapeutic effect on rheumatism, 668

Tonsillectomy—Continued
tonsil knife and adenotome for use with head in hyperextended position, *934
treatment of follicular focal infection from standpoint of internist, with reference to electrocoagulation of tonsils, 336
Tonsillitis: See under Tonsils
Tonsils, Abscess: See Abscess, peritonsillar
and adenoids, 504, *653
chronic tonsillitis in secondary syphilis; differential diagnosis from diphtheria and Vincent's infection; report of 23 cases, 134
complications, *660
fusospirochetal organisms and tonsillitis; bismuth therapy of tonsillitis, *587
intracranial complications of tonsillar origin with reference to their incidence and pathogenesis, *530
papilloma of tonsil and uvula, *810
postoperative angina, 505
primary lymphosarcoma of, 815
role of tonsils and adenoids in scarlet fever, 342
tumors, benign and malignant, *662
Ulcer: See Fusospirochetosis
x-ray treatment in infections of tonsils and pharynx, 504
Trachea, acute laryngotracheitis treated by intubation, tracheotomy and sulfapyridine, 992
giant cell tumor of, *962
grippal tracheobronchitis and its treatment, 337
importance of control of pulmonary lesion when tuberculous tracheobronchitis coexists, *408
present status in treatment of acute obstructive infections of larynx, trachea and bronchi, 669
results of treatment of tuberculosis of trachea and bronchi, 816
tracheobronchial tuberculosis, *945
Tracheotomy, acute laryngotracheobronchitis in newborn infant; report of case with recovery following tracheotomy, *423
Trauma, traumatic perforation of right frontal sinus with pneumocephalus and brain abscess; report of case, 349
Tremble, G. E.: Case of ulcerative pharyngitis with impaired myeloid activity following use of sulfanilamide for gonorrheal infection, 996
Frontal and ethmoid sinusitis treated by external frontal ethmoidectomy, 996
Sphenoid sinus; safe and simple method of irrigation, *755
Trowbridge, B. C.: Electroaudiography; analysis and interpretation of audiogram, *899
Local use of sulfanilamide and its derivatives in ear, nose and throat, *740
Tuberculosis: See also under names of organs, regions and diseases, as Bronchi; Ear; Larynx; Osteoperiostitis; Otitis Media; Trachea; etc.
importance of control of pulmonary lesion when tuberculous tracheobronchitis coexists, *408
pulmonary; laryngeal tuberculosis; study of 500 cases of pulmonary tuberculosis, with résumé based on 28 years' experience, 682
Tucker, G.: Interrelations, diagnosis and treatment of benign and malignant tumors of larynx, with reference to prevention and cure of laryngeal cancer, 677
Tuta, J. A.: Neuroepithelioma in temporal bone; invasion of petrous and squamous portions, with extension into middle and posterior cranial fossae, *745
Tumors: See Adamantinoma; Adenoma: Cancer; Chondroma; Epithelioma; Fibroma; Hemangioma; Leiomyosarcoma; Lipoma; Melanoma; Papilloma; and under names of organs and regions, as Brain; Bronchi; Ear; Esophagus; Larynx; Pharynx; Temporal Bone; Tonsils; Trachea: etc.
Tuning Forks: See Hearing, tests
Tympanic Membrane: See Ear, tympanic membrane

Ulcers: See also under names of organs and regions
surgical management of postradiation scars and ulcers, 339
Tonsillar: See Fusospirochetosis
Uvula, papilloma of tonsil and uvula, *810

Vaccine Therapy: See Sinuses, Nasal
Veins: See also Thrombosis
acute suppurative otitis media with thrombosis of lateral sinus in child, treated by mastoidectomy, ligation of internal jugular vein and sulfapyridine, 993
Vertigo, aural; case for diagnosis: (1) labyrinthitis?; (2) Ménière's syndrome? (3) subdural abscess? 347
aural; evaluation of Ménière syndrome, 980
aural; labyrinthine dropsy and Ménière's disease, *853
aural; nature and treatment of Ménière's disease; further clinical observation, 502
aural; surgical treatment of Ménière's disease, 667
Vestibular Apparatus: See Ear; Labyrinth
daVinci, Leonardo; contribution to laryngology, rhinology and phonetics, 836
Vinson, P. P.: Lipoma of left main bronchus; report of case and review of literature, *868
Vitamins, A; ear in experimental vitamin A deficiency, 981
A; utilization of vitamin A added to mineral oil, 818
Vocal Cords: See also Larynx: Voice
Paralysis: See Larynx, paralysis
physiology in phonation and respiration, *355
subcutaneous hematoma of neck following vocal strain, 506
Voice: See also Vocal Cords
Leonardo da Vinci's contribution to laryngology, rhinology and phonetics, 836
physiology of vocal cords in phonation and respiration, *355
tonsillectomy and its effect on singing voice, *915

War: See also Air Raids; Aviation and Aviators; Medicine, Military; Wounds; etc.
acoustic trauma in war and in peace, 834
and hearing, *471
malingering and neuro-otologic considerations in combat, 173
prevention and treatment of acoustic trauma in war and in civil life, 172
Water, role of water balance in clinical manifestations of allergy, 672
study of changes in temperature and water vapor content of respired air in nasal cavity, 820
Waugh, T. R.: Hematologic aspects of chemotherapy, 990
Weaver, D. F.: Benign encapsulated fibroma of larynx, *288
Weinstein, S.: Otitic bacterial meningitis, *53
White, L. E.: Chronic suppurative otitis media on right, with labyrinthitis following removal of aural polyp, 344
Whitehead, R. W.: Chemotherapy of tuberculous otitis media, 158
Wilkerson, W. W., Jr.: Chordoma, *925
Witting, E. G.: Analysis of speech characteristics in deafened children, with observations on methods of training, 160
Woodruff, G. H.: Endaural mastoidectomy; experience in series of 76 cases, *777
Woodward, F.: Use of temporary inexpensive bite block to determine relationship of closed bite and symptoms referable to temporomandibular joint, 156
Wounds, gunshot, of frontal and temporal bones, 831
local use of sulfanilamide powder and hydrogen peroxide in wound infections, 986

Zerffi, W. A. C.: Tonsillectomy and its effect on singing voice, *915

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CONTENTS

PAGE

Use of Radium in Treatment of Deafness by Irradiation. Ernest B. Emerson Jr., M.D.; Andrew H. Dowdy, M.D., and Clyde A. Heatly, M.D., Rochester, N. Y. 845

Labyrinthine Dropsy and Ménière's Disease. J. R. Lindsay, M.D., Chicago 853

Lipoma of Left Main Bronchus: Report of a Case and Review of Literature. Porter P. Vinson, M.D., and Williams E. Pembleton, M.D., Richmond, Va. 868

Studies on Pupils of the Pennsylvania School for the Deaf: IV. Mechanism of Inheritance of Deafness. Walter Hughson, M.D., and Antonio Ciocco, Sc.D., Abington, Pa., and C. E. Palmer, M.D., Bethesda, Md. 871

Estrogenic Implants in the Treatment of Atrophic Rhinitis. Lloyd K. Rosenvold, M.D., Los Angeles 883

Hypodynamia Laryngis. Milton Sills Lloyd, M.D., New York 893

Electroaudiography: Analysis and Interpretation of the Audiogram. Barnard C. Trowbridge, M.D., Kansas City, Mo. 899

Tonsillectomy and Its Effect on the Singing Voice. William A. C. Zerffi, New York 915

Experimental Hypersensitivity of Mucous Membranes of the Upper Respiratory Tract. Ira Frank, M.D.; Margery Blahd, M.D., and Katharine M. Howell, M.D., Chicago 918

Case Reports:

Radical Mastoidectomy with Complete Recovery in a Case of Chronic Lymphatic Leukemia. Arthur J. Cracovaner, M.D., and Carl Reich, M.D., New York 922

Chordoma. W. W. Wilkerson Jr., M.D., Nashville, Tenn. 925

Adamantinoma of the Floor of the Right Nostril: Occurrence in a Child Aged Five Years. Alfred B. Berkove, M.D., Oakland, Calif. 928

Chancre of the Nasal Septum. Arthur J. Cracovaner, M.D., New York 932

Clinical Notes; New Instruments and Technics:

Tonsil Knife and Adenotome for Use with Head in the Hyperextended Position. George E. Shambaugh Jr., M.D., Chicago ... 934

Progress in Otolaryngology. Summaries of the American and British Bibliographic Material Available in the Field of Otolaryngology: Peroral Endoscopy. Louis H. Clerf, M.D., and F. Johnson Putney, M.D., Philadelphia 936

Abstracts from Current Literature 980

Society Transactions:

Montreal Medico-Chirurgical Society, Section of Otolaryngology .. 988

Directory of Otolaryngologic Societies 998

General Index 999